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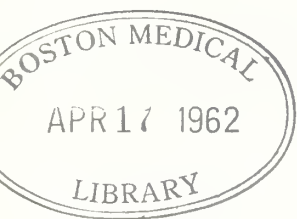
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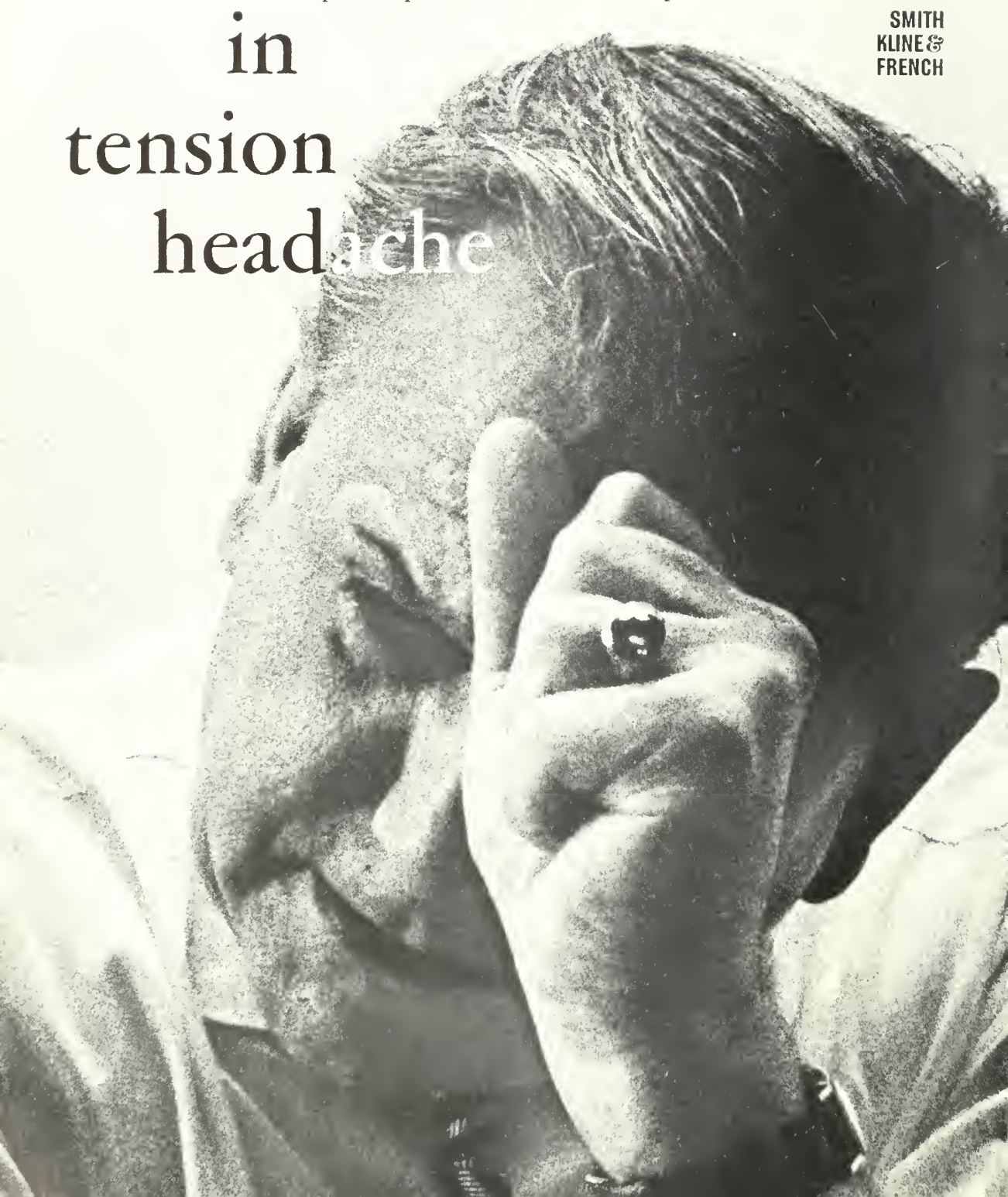
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Needs for Further Progress in Venereal Disease Control

E. GURNEY CLARK, M.D., Dr. P.H.

New York City

FURTHER PROGRESS IN venereal disease control will be inhibited unless certain specific needs are considered and fulfilled. These needs have been determined recently by local and national studies. One of these studies is the annual review of current venereal disease control problems studied by means of a questionnaire sent to all states and territories, all cities in the United States with populations over 100,000, and to the District of Columbia. In fiscal 1958, replies were received from 49 states, 3 territories, 95 out of 107 cities, and the District of Columbia. The results are presented in the sixth annual joint statement, *Today's Venereal Disease Control Problem*, recently released by the Association of State and Territorial Health Officers, the American Venereal Disease Association, and the American Social Hygiene Association.¹

The number of cases of syphilis and gonorrhea and the rates per 100,000 population reported by state health departments from 1941 to 1956 were shown in 1958 in the paper published in THE JOURNAL-LANCET entitled "Years of Progress in Venereal Disease Control."² Considerable progress has been made, but venereal disease is still a major health hazard. In fiscal 1958, reported cases of primary and secondary syphilis increased in the United States to 6,685 from 6,283 in 1957. Increases occurred in 23 states and 26 of the surveyed cities. The number of cases of

early latent syphilis reported in the United States as a whole in 1958 was 19,308, which was less than the 20,346 cases in 1957, but there were significant increases in 16 states and 20 cities. Reported cases of late and late latent syphilis in fiscal 1957 numbered 100,514, and, in 1958, there were 92,781 cases reported. This is a national decline in reported cases, but there were rises in 19 states and 23 cities. Reported gonorrhea rose from 216,476 cases in 1957 to 220,191 in 1958.

Since the joint statement was published in February 1959, additional information on infectious syphilis has become available from state and local health departments and shows a 22.8 per cent increase in reported primary and secondary syphilis for fiscal 1959 as compared with fiscal 1958. In the same period, gonorrhea has increased 7.8 per cent.

The increases in infectious syphilis have been noted in 28 states and in gonorrhea in 39 states among both races and among both clinic patients and those under the care of private physicians. Recent data also show an increase in early latent syphilis for the country as a whole.

These alarming trends have had some good effect, however, and have stimulated venereal disease control personnel to focus more attention on the control of infectious syphilis. Complacency concerning syphilis has been replaced in many quarters by a real concern to achieve once and for all the goal of practical eradication in this country. The term "practical eradication" is interpreted to mean the reduction of the incidence of syphilis to the level of occurrence of such diseases as malaria.

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To attain this goal, control efforts in most states now place special emphasis on the concept that every case of early syphilis is an emergency and must be reported and that each of these patients must be interviewed for sex contacts by a trained interviewer from the health department. This same intensive case finding work is being applied to patients of private physicians as well as clinic cases. Particular effort is being made to urge private physicians to report every case and have each patient interviewed for contacts. The private physician aspects of venereal disease control will be more fully discussed in a later section of this paper.

ADDITIONAL INFORMATION ON THE ANNUAL JOINT STATEMENT

Venereal disease increased among young people in the fiscal year 1958, when 22 states and 31 cities of 100,000 population and over reported increases among the 15 to 19 age group, and 12 states and 13 cities reported increases among the 10 to 14 age group. This represents a significantly higher number of states and cities reporting increases in venereal disease among those of teen-age than in fiscal 1957, when 14 states and 19 cities reported increases in the 11 to 19 age group and, in fiscal 1956, when 11 states and 18 cities also reported increases in the teen-age group.

The number of states and cities reporting venereal disease outbreaks continues to increase—24 states and 21 cities in fiscal 1958. This is more than in fiscal 1957, when 20 states and 17 cities reported such outbreaks. An outbreak is a cluster of cases which, by epidemiologic investigation, have a common source and occur within a relatively short period of time.

It is obvious that rates for the country as a whole do not define the extent of the problem in individual states and cities. In addition to this, it is obvious from the reports of 34 states, 1 territory, and 47 cities that the rates for large areas tend to conceal high prevalence rates in small areas within them.

In 34 states, 2 territories, and 25 cities, military personnel created special demands on local venereal disease programs. Twenty states, 1 territory, and 8 cities reported that interstate migrant labor created special demands on local programs. Ten states, 1 territory, and 10 cities found that international migrant labor creates special demands.

Other persons with venereal diseases are migrant also. In 13 states and 18 cities, 5 to 9 per cent of the sexual contacts of patients were out-

side the state or city in which the patients resided. Health departments of 21 states and 11 cities reported that they received outside information which enabled them to find and treat infections locally in persons who were infected away from the departments' own jurisdiction. One state reported receiving such information from 25 other states and foreign countries. Thus, intercity, interstate, and international control measures must be considered.

Over 22,000,000 people in the United States live in areas which health departments regard as inadequately protected against venereal disease. Health departments in 35 states consider that 79 cities, 426 counties, and 9 other areas with combined populations totaling 22,708,411 are not well covered. Health departments in 8 cities consider that 30 census tracts, 3 election districts, and 10 other areas with combined populations totaling 1,789,000 are not well covered.

Some physicians are provided with contact interviewing services by their local health departments. In 37 states, 2 territories, and 57 cities, a *small* percentage of the patients of private physicians are interviewed and their contacts sought.

Shortage of personnel is a problem. Seven states and 4 cities do not have sufficient investigative personnel to interview all reported cases of primary and secondary syphilis and search for their contacts, and 17 states, 1 territory, and 9 cities are unable to investigate the contacts of all reported cases of early latent syphilis in their areas. States and cities report a real need for federal support of state and local programs. Forty-one states, 2 territories, and 59 cities reported that they could not carry out their venereal disease case-finding efforts without federal assistance.

Based on the data from this national survey, the following recommendations were made:

1. There should be a minimal federal appropriation for venereal disease control of \$5.8 million in fiscal 1960, with at least \$2.8 million for grants to states. This would be an increase of \$400,000 over the present appropriation.

2. State and local health departments should be encouraged to plan and make epidemiologic case-finding services available to the private physicians and to seek such additional funds as are needed to implement their plans.

3. A program of social and health education should be directed toward the teen-age group, and the White House Conference on Children and Youth in 1960 should be encouraged to give special consideration to this teen-age venereal disease problem.

ADDITIONAL INFORMATION ON
CURRENT VENEREAL DISEASE PROBLEMS

No one will deny that much progress has been made in venereal disease control. The basic epidemiologic facts from various parts of the United States reveal that the problem is still one of considerable public health importance, and it may become more important if vigilance is relaxed and if current needs are not met.

Due to the fact that, in 1957, there were 19 cases of latent and late syphilis treated and reported for every case of infectious syphilis reported, it is obvious that every year the majority of new cases have not been discovered. The statisticians of venereal disease branch of the Communicable Disease Center of the Public Health Service have estimated that, excluding armed forces personnel, the annual incidence of syphilis is about 60,000 cases. Since the annual average number of *reported* cases of infectious syphilis over the past five years is about 6,500, probably about 53,500 each year are not discovered or not reported. For example, in one large city of the United States in 1953, a study was made of case reporting by private physicians. Information was requested from all physicians, and 75 per cent responded to the questions. The respondents indicated that they had seen 3,112 cases of syphilis during that period. A review of the cases reported to the health department showed that only 753, or 24.2 per cent, of these cases had been reported. Thus, in this *one* urban area of the United States, 2,359 cases were seen by physicians and not reported in 1953.

Unless increased efforts are made to discover and treat syphilis, it is possible that 25 per cent of the untreated syphilitic patients may become infectious again. Cardiovascular disease may develop in 10 per cent, mental health defects in 7 per cent, and more than 10 per cent may die as a result of syphilis.^{3,4} Every effort should be made to find these cases as early as possible to prevent illness and reduce financial obligations of local and national areas.

Other information from the venereal disease branch of the Communicable Disease Center shows that syphilis, a preventable disease, is very costly unless early case finding and treatment are carried out. In a recent scientific report by Dr. William J. Brown, chief of the venereal disease branch, the following important data were presented. Syphilis still kills a minimum of about 4,000 persons a year (these are the known cases). It costs \$12 million a year to care for the blind syphilitic patients in this country. Loss of income by men with advanced syphilis is

estimated at \$100 million per year. It costs \$46 million a year for hospitalization of 32,000 syphilitic psychotic patients.

OUTSTANDING NEEDS

In addition to increasing the usual routine methods of venereal disease control, two very important procedures should be promoted and followed in order to find additional cases and further reduce the spread of venereal disease: (1) more private physicians—health department collaboration (*this is essential*) and (2) cluster testing for case finding (very productive).

In the address given by Dr. Brown,⁵ a most interesting analogy was made which illustrates one of the great needs. Just as cancer may metastasize throughout the body, venereal disease spreads throughout the community. Syphilis is a "community malignancy." Just as cancer cannot be controlled without searching in all cases for the disease beyond the primary site in the body, syphilis cannot be controlled without searching for all early cases in persons who have contacted the individual known to have syphilis. Contact tracing is essential for adequate case finding, and cases *must be found* to be treated, and syphilis *must be treated* to prevent syphilitic cardiovascular disease, central nervous system syphilis, and premature death. The following quotations taken from Brown's⁵ address given at a private physician seminar clarify the foregoing, "As patients are diagnosed, they are interviewed and asked to name those persons to whom they were exposed sexually since the beginning of the incubation period up to the time when treatment renders them noninfectious. These 'contacts' are then located, examined, and, if necessary, treated, all in the strictest confidence. In this way, 160,000, or about half, of the known venereally infected persons are interviewed annually in this country and, through them, an additional 150,000 sex contacts are located, examined, and, if necessary, treated.

"If we are to achieve our goal of the practical eradication of syphilis in this country, every case of syphilis will have to be reported, each patient will have to be interviewed, all contacts will have to be checked for syphilis, and all infected persons will have to be treated.

"In the United States, in a five-year period, 1954 through 1958, 14,428 cases of primary and secondary syphilis were reported by private physicians, and, of this total, only 2,400 patients were made available for contact interviewing by trained interviewers. This is only 16.6 per cent of the patients reported. From all other sources, 19,521 cases of primary and secondary syphilis

were reported, and 16,959 of the patients, or 87 per cent, were interviewed for sex contacts by trained health department interviewers." The 2,400 patients of private physicians who were interviewed led to 8,016 contacts. Thus, the 12,028 patients who were not interviewed probably would have yielded 40,173 additional contacts, many of whom would have been infected and could have been treated to prevent further disability and spread.

Fortunately, there are a few areas in the United States in which private physicians are working with their health departments in a cooperative plan to control syphilis. Dr. Brown mentioned one state in which a detailed cooperative contact investigation program had been in operation with a high degree of success. More than 10 times as many cases of syphilis were reported by the private physicians in this state in the three years following the initiation of the cooperative program than had been reported in the previous three years. Since such a program proved successful in one state, there is no reason why it cannot be used successfully in every state.

The importance of this need was emphasized in 1943, the year of highest syphilis rate, by Joseph Earl Moore, one of the most prominent venereal disease specialists in this country.⁶ "Organized case finding by routine serologic testing and the epidemiologic approach offers a solution of many of the difficulties encountered in getting patients under treatment. If they are generally applied, the demand for treatment facilities would be from 2 to 3 times greater than at the present."

The basic concepts of the epidemiology of syphilis with particular reference to contact investigation was reviewed in 1948 in a brief paper with 96 references on the subject.⁷ The following quotation from this paper is applicable today: "Patients are willing, under proper circumstances, to divulge information concerning these intimate contacts, and the contacts, in turn, are willing to submit to medical examination if they are approached in a suitable manner. A patient can be expected to withhold information about his intimate contacts because he may be ignorant of the potentialities of the disease. The physician, on the other hand, is fully aware of these potentialities, and it is his obligation to inform the patient of the dangers of the disease to himself and to his contacts. Failure to participate in this manner in contact investigation is failure to assume a medical responsibility. It has been shown that successful participation of a private physician in this phase of syphilis control is practicable."

It is obvious that contact interviewing by the

private physician is a time-consuming contribution to venereal disease control, and contact tracing requires more time than any physician has available. For these reasons, many private physicians, in order to improve venereal disease control, will wish to request health department personnel to do this important work for them. In the areas in which private physician participation has been most productive, health departments have supplied continuous investigation services. This fact is the substance of a 10-state evaluation and demonstration proposal that the American Social Hygiene Association presented to the Congressional Committee in April 1959.

Venereal disease in the United States will never be controlled independently of the private physician.⁸ Between 1953 and 1955, private physicians in Georgia had reported a total of 664 cases of syphilis. In 1956, the state introduced a private physician reporting program based upon three principles: (1) personal contact with physicians, (2) a reporting form that was clear and required practically none of the busy physicians' time, and (3) special service at every contact point in order to make the plan work. The almost unprecedented result was that, between 1956 and 1958, Georgia's physicians reported 6,940 cases, well over half of all cases in the state. These Georgia data show conclusively that private physicians are willing to cooperate and that, when they do, the control effort becomes tremendously more effective.

The second important procedure which will add significantly to venereal disease control is the increased use of "cluster testing." Cluster testing is a new case-finding technique which is an extension of the standard contact investigation process. Patients with infectious syphilis are interviewed for sexual contacts as usual and are also asked to name other persons, designated as "named suspects," of either sex who move in the same sociosexual environment. In addition, associates of patients, contacts, and suspects are blood tested. Associates include neighbors, fellow employees, and others who have not been specifically named as contacts or suspects.

To quote again from Dr. Brown:⁵ "It has now been shown that in addition to sex contacts, the group of convivial associates of infected persons also includes many infected and noninfected persons. Recently, venereal disease specialists have begun to trace the disease through these chains of association as well as through the chains of sex contacts and, in this way, have almost doubled the number of infectious syphilis cases which could be traced from one known infected person."

In the Georgia demonstration study of the cluster testing technic in fiscal year 1958, the technic was applied to 515 patients with primary and secondary syphilis. The 2,391 sexual contacts of these 515 patients who were examined yielded 227 cases of primary and secondary syphilis, or 44 cases per 100 patients. This yield from contacts is above average. By extending the investigation process from contacts to suspects and associates, an additional 8,285 suspects were examined, and 94 more cases of primary and secondary syphilis were discovered—an additional 18 cases per 100 primary and secondary patients. Thus, the yield of primary and secondary cases discovered through this investigation process was raised from 44 per 100 patients from sexual contacts alone to 62 per 100 patients, an increase in yield of infectious syphilis of over 40 per cent.

In addition, 136 patients in other stages of syphilis were discovered among the suspects and associates who would not have been found if the cluster testing technic had not been employed.

More recent data evaluating cluster testing show even higher yields of newly discovered patients with infectious syphilis.

CONCLUSIONS

The venereal disease problem has changed significantly both in extent and in significance. The significance of the problem no longer relates primarily to the province of prostitution; no longer is easy-to-get penicillin considered substitute for other control efforts; no longer is the spread of venereal disease infection considered a simple chain; and no longer can public health workers consider that control can be effective by their own efforts alone.

Great progress has been made in venereal dis-

ease control, but the extent of the problem is still of considerable public health importance and further progress will be impaired unless outstanding needs are met. The most outstanding need is additional case finding efforts. Venereal disease in the United States will never be controlled without private physician collaboration in community case finding efforts.

If carried out, the following recommendations will do much to aid in the control of venereal disease.

1. The practical eradication of syphilis in the United States should be our goal.

2. Intensified case-finding efforts should be focused on the control of infectious syphilis. Every case should be considered an emergency, be reported, and each patient be interviewed for sex contacts by a trained health department interviewer.

3. Private physicians should be encouraged to become an integral part of the over-all syphilis control program. Their participation is essential for further progress against syphilis.

4. Special studies of venereal disease problems among teen-agers should be continued so that new epidemiologic techniques can be applied to this group.

5. Intensive venereal disease informational and educational programs should be carried out.

6. Further research should be encouraged on the perplexing problems of diagnosis of gonorrhea, particularly in the female, so that some progress can be made against this venereal disease in the future.

Copies of the annual report, *Today's Venereal Disease Control Problem*, are available each year from the American Social Hygiene Association, 1790 Broadway, New York City.

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Blunt Trauma to the Abdomen

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TRAUMA EXISTS among us as an endemic disease which occasionally assumes epidemic proportions. Available data¹ indicate that, during the course of the year, nearly one third of our population incur some form of injury requiring medical attention or absence from work for at least a day. Trauma is a major cause of death, surpassed in importance for the entire population only by cardiovascular disease and cancer (tables 1 and 2). It is first among the causes of death between the ages of 5 and 25 years. Among the elderly, while not the leading cause of death, fatal trauma is far more prevalent than in the younger age groups (figure 1). In sharp contrast to developments in many of the infectious diseases, the death rate from accidents has continued through the years with very little improvement (figure 2).

Much has been written and said about the management of open and penetrating wounds of the abdomen. The experience in two world wars and in the Korean action has demonstrated the safe approach to their management. Blunt wounds, especially those of the abdomen, remain treacherous because serious intra-abdominal injuries are often masked by wounds which may be more obvious but are less likely to be fatal. Furthermore, the physician is too often led astray by the fact that, during their early stages, fatal intra-abdominal lesions can masquerade as trivial or nonexistent. Thus, diagnosis becomes a consideration of first importance.²⁻⁴

DIAGNOSIS OF INTRA-ABDOMINAL INJURY AFTER BLUNT TRAUMA

The obvious case. The signs of intra-abdominal injury, when typical and fully developed, are easily recognized. They consist of tenderness and spasm due to irritation of the parietal peritoneum by blood or by the contents of the gastro-

intestinal tract and urinary bladder; peritoneal bulging and shifting dullness indicative of an accumulation of fluid, usually blood, within the peritoneal cavity; referred pain of diaphragmatic irritation to the ipsilateral shoulder; pneumoperitoneum when the stomach or colon are ruptured; respiratory embarrassment and high intestinal obstruction with diaphragmatic rupture and herniation of abdominal viscera into the thorax and; finally, the signs and symptoms of shock.

When these findings are present, the surgeon has no difficulty in diagnosing an intra-abdominal injury, and the course of action is clear. But, as already mentioned, these conditions are treacherous because, only too often, fatal intra-abdominal injury may exist with no abnormality detectable early after injury. Therefore, the safe management of the patient with an occult injury after blunt abdominal trauma demands the most careful evaluation of the details of history and physical examination.

The patient with an occult intra-abdominal injury. Sad experience has taught that the safest policy to follow is to suspect immediately that any patient who has incurred blunt abdominal trauma has a serious injury until proved otherwise. The suspicion is strengthened by the following considerations:

1. *Characteristics of the injury.* Blunt abdominal violence which renders the patient temporarily unconscious and is followed by nausea, pallor, or "cold sweat" or throws the patient off his feet and against a solid object must be suspected of having caused serious intra-abdominal injury.

2. *Characteristics of the patient.* While it is true that some individuals welcome the slightest pretext for admission to a hospital, they are exceptions, and the majority of people prefer to remain away unless they feel that something is radically wrong with them. The physician will do well to keep this in mind, for the patient can sometimes sense impending catastrophe despite the absence of overt physical signs. As to the physical examination, scratches and lacerations may be of no significance with regard to internal

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Paper presented at a symposium on "Automobile Trauma" at Methodist Hospital, St. Louis Park, Minnesota, November 14, 1959.

TABLE 1
SIX MOST COMMON CAUSES OF DEATH BY AGE GROUP
(Statistical Abstract of the United States, 1959)

Causes of death	Age group (year inclusively)				
	5-14	15-24°	25-44	45-64	65+
Accidents	I	I	II	IV	V
Malignant disease (not leukemia)	II	II	III	II	III
Tuberculosis	—	IV	IV	V	—
Suicide	—	V	V	VI	—
Heart disease	—	III	I	I	I
Cerebral hemorrhage and thrombosis	—	—	VI	III	II
Hypertension and arteriosclerosis	—	—	—	—	IV
Pneumonia	VI	—	—	—	VI

° Nephritis is No. VI among causes of death in 15-24 age group.

TABLE 2
SIX MOST COMMON CAUSES OF DEATH BY AGE GROUP
(Statistical Abstract of the United States, 1959)

Causes of death	Age group (year inclusively)				
	0-4°	5-14	15-24	25-44	45-64
Pneumonia	IV	VI	—	—	—
Congenital malformations	V	IV	—	—	—
Accidents	VI	I	I	II	IV
Leukemia	—	III	—	—	—
Other malignant disease	—	II	II	III	II
Acute poliomyelitis	—	V	—	—	—

° First, second, and third places in the 0-4 age group are occupied by immaturity, postnatal asphyxia, and birth injury, respectively.

injury, but ecchymosis, hematoma, and contusion of the abdominal wall and trunk must be viewed with alarm. Fractures of the lower ribs on either side should make the surgeon very suspicious of injuries to the diaphragm, kidney, liver, and spleen. The patient who has incurred blunt abdominal trauma and simply does not look well must be suspected of having serious injury, even if no specific diagnosis can be made at the time.

The fatal error of sending the patient away with instructions to return "if he feels bad" or "if he feels worse" must be avoided. Examples are commonplace of patients who have died on such prescribed return trips to the same or other hospitals. Laymen cannot be expected to render sophisticated judgment as to when the situation is deteriorating to the extent that return to the hospital is indicated. The following is a case in point:

DEATHS DUE TO ACCIDENTS PER 100,000 WHITE POPULATION
(UNITED STATES, 1950)

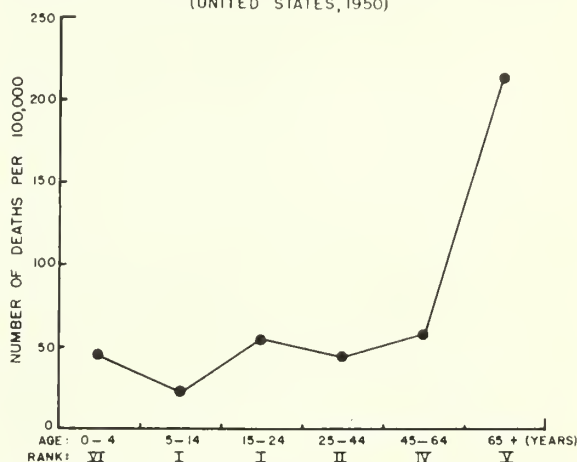


Fig. 1. Deaths from accidents per 100,000 population (ordinate), related to age groups (abscissa). Roman numerals indicate, by age group, the relative position of accidents among the leading causes of death for that age group. (Adapted from Health Statistics, from the U. S. National Health Survey, U. S. Department of Health, Education and Welfare.)

DEATH RATE PER 100,000 POPULATION

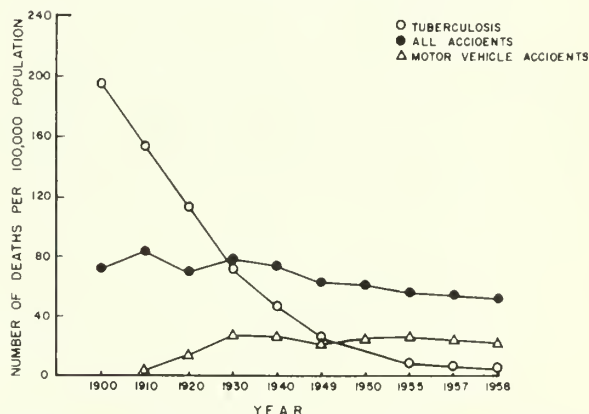


Fig. 2. Deaths per 100,000 population, comparing tuberculosis, all accidents, and motor vehicle accidents. (Statistical Abstracts of the United States, 1959. Government Printing Office, Washington, D. C.)

CASE REPORT

A.B., a 3-year-old boy, fell against a bedpost while riding his tricycle in the bedroom at about 9:00 P.M. His mother thought he whimpered more than usual after this fall, and she promptly brought him to our emergency room. When seen, no abnormality could be detected, and the child wasn't even registered so that the mother would not be charged a fee. Instead, she was advised to take the child home but to bring him back if he showed any unusual behavior. During the night, the child did not sleep well, cried, and vomited twice. However, these indications were not considered suffi-

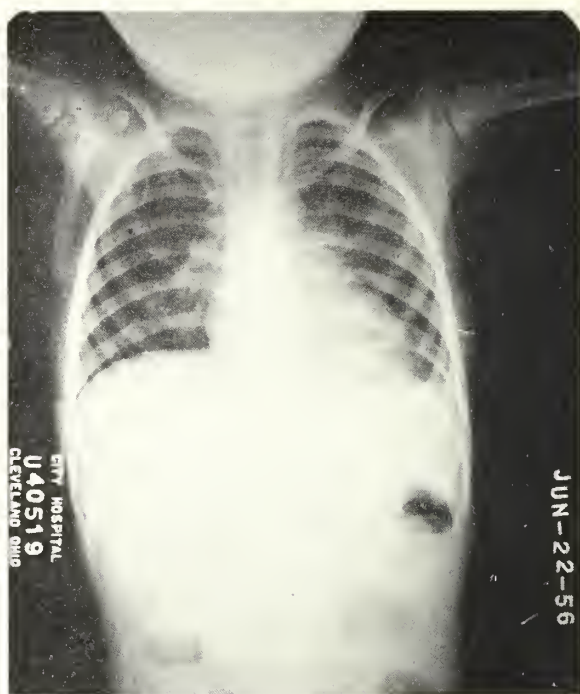


Fig. 3. Plain roentgenogram of the abdomen in patient A.B. (*Left*) Note signs of ileus without air beneath diaphragm (*right*).

ciently serious to bring the child back. During the day, he became progressively worse, and Mrs. B. brought him to the emergency room at 5:30 p.m., some twenty hours after injury. He now had obvious diffuse peritonitis with paralytic ileus (figure 3). At operation, he was found to have a perforation of the terminal ileum on the antimesenteric border. The edge of the perforation was excised, and the defect was repaired. Histologic examination of the resected ileal tissue showed peritonitis but no suggestion of a primary intestinal lesion.

This experience again taught us that very little violence is needed to cause traumatic perforation of the intestine and that even an attentive mother cannot be expected to exercise adequate medical judgment.

The astute clinician finds additional characteristics to strengthen the suspicion of intra-abdominal injury following blunt trauma to the abdomen. A history of antecedent hydronephrosis, of course, makes damage to the kidney almost certain. We have seen a patient with a renal neoplasm which ruptured after trauma so trivial that its role was seriously questioned. Structural scoliosis displaces the kidney, pancreas, and liver. In such patients, these organs are more susceptible to trauma than they are in normal individuals. The "malaria spleen" and the spleen during or shortly after infectious mononucleosis are notoriously prone to rupture, even spontane-

ously. The large liver of early cirrhosis or the engorged liver of the early postprandium are more likely to be injured than the normal or "resting liver."

3. Aids to clinical diagnosis.

A. *Examination of the urine.* Gross blood in the urine is indicative of injury to the kidney and its excretory passages. Localization of the injury and information regarding its characteristics can be obtained by urologic roentgenographic examination. Emergency excretory rograms can be made when hypotension is absent or has been corrected. If the patient is unable to void, an indwelling catheter is placed in the urethra. Cystograms are made to delineate possible injury to the bladder.

B. *Examination of the blood.* Patients who have incurred trauma often present elevations of the white blood cell count with predominance of the polymorphonuclear series. This occurs with fractures, for instance, as well as with intra-abdominal injuries and, therefore, cannot be taken as specific. Determination of the hematocrit is important as a baseline for subsequent comparison, but significant hemodilution should not have had a chance to occur before the decision to operate is made on the basis of other findings. A high normal value of the hematocrit,

or actual hemoconcentration, should make one suspicious of injury to an abdominal viscus. Retroperitoneal rupture of the intestine should be suspected when grossly abnormal findings are not elicited on physical examination of the abdomen but leukocytosis and hemoconcentration persist.

C. *Roentgenologic examination.* At least one plain roentgenogram of the abdomen should be obtained in all patients who are suspected of having intra-abdominal injury. A recumbent supine position should first be examined, and other views can be obtained depending upon the findings in the first. These initial films are helpful not only as aids in establishing an immediate diagnosis but also for comparison with subsequent films which may be taken later.

D. *Paracentesis.* We have been unenthusiastic about this procedure not because it is dangerous but because it should be unnecessary for diagnosis and, in fact, may be misleading. Thus, in the study reported by Williams and Zollinger,¹ in which 32 patients were so examined, evidence for intra-abdominal bleeding was revealed in 25. However, there were 2 false positive tests and 3 false negative paracenteses in patients with rupture of the spleen.

DIAGNOSIS OF INTRA-ABDOMINAL INJURY AND INDICATIONS FOR OPERATIVE INTERVENTION

The decision for operative intervention in patients who have incurred blunt abdominal trauma is not predicated by absolute and specific diagnosis. The strong suspicion that one or more of the vital structures within the abdomen and pelvis has been damaged is sufficient indication for surgical intervention. Such presumptive diagnosis is made upon the following observations:

1. *Evidences of parietal peritoneal irritation*—tenderness, spasm, and rigidity. These are indications of bleeding within the abdomen or peritonitis from contamination of the peritoneum by gastrointestinal contents or both.

2. *Accumulation of fluid within the abdomen*—shifting dullness. This finding suggests massive bleeding within the peritoneal cavity. Subperitoneal bleeding may be palpable or percussible but is not shifting.

3. *Adynamic ileus.* This may result from intra-abdominal hemorrhage or from rupture of the gastrointestinal tract, causing peritonitis. More deceptive, however, is retroperitoneal perforation of the intestine, which may manifest itself only by ileus, fever, and prostration.

4. *Shift of the trachea and mediastinum with cardiorespiratory embarrassment.* Diagnosis of rupture of the diaphragm, usually the left,

should be suspected with or without concomitant intra-abdominal injury. Such injury to the diaphragm has been missed because it was not suspected. Roentgenograms are used for definitive diagnosis (figure 4).

5. *Development of fever during the first few hours of observation.* In the absence of other abdominal findings, fever should make one strongly suspicious of retroperitoneal rupture of the intestine. Careful and repeated physical and roentgenographic examinations of the abdomen are very helpful.

6. *Roentgenographic demonstration of increasing size of a space-occupying abdominal mass.* Such increasing density demonstrable on repeated abdominal roentgenograms is often the principal indication of hemorrhage from a ruptured spleen.

7. *Development of clinical shock.* When the patient arrives in shock or when shock develops while he is under observation, serious hemorrhage must be suspected. In this connection, two points must be borne in mind. First, the prominent clinical features of shock are thirst, pallor, and coldness of the face, hands, and feet. These symptoms develop before there necessarily is a drop in the blood pressure, which does not fall until the compensatory mechanisms fail. However, the pulse rate almost always shows a progressive increase as one of the compensatory mechanisms. Second, the physician must remember that, while it is a truism that "shock must be corrected before operation is undertaken," operation sometimes must be undertaken as the very means of correcting the shock. Such is the case when there is massive bleeding from a major vessel. Judicious delay is tolerable only when there is good evidence that the cardiovascular status of the patient is improving under the therapy adopted. It follows from these considerations that operation after blunt abdominal trauma is undertaken when a diagnosis of intra-abdominal injury is reached but without necessarily a more specific diagnosis. For this reason, great care must be exercised in the choice of the incision. In our experience, a long right or left paramedian incision permits thorough examination of the abdomen and pelvis. After having found one or more lesions, the surgeon must not be trapped into missing associated injuries in the interest of a small incision.

ORGANS COMMONLY INJURED IN BLUNT ABDOMINAL TRAUMA

In decreasing order of frequency, the organs commonly involved in damage after blunt abdominal trauma are the kidney, the spleen, the

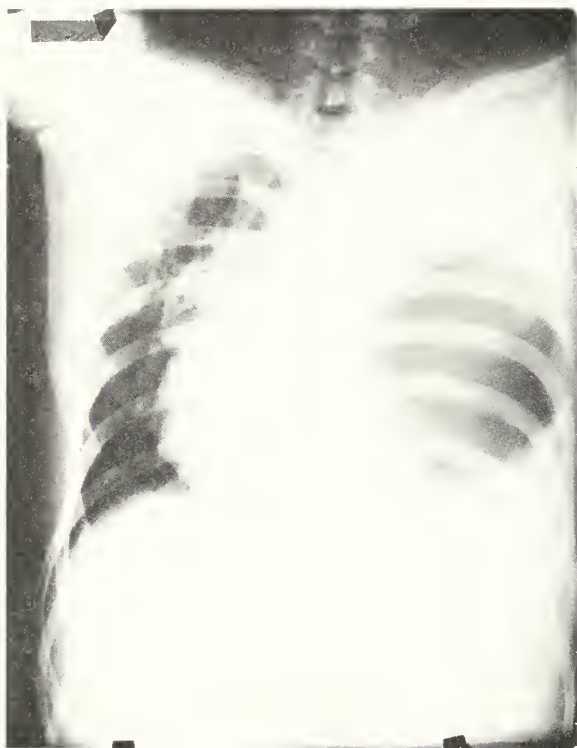


Fig. 4. Rupture of diaphragm. (*Left*) Rupture of left leaf of diaphragm. Note displacement of mediastinum to right. Gas in left side of thorax lies in herniated stomach and colon. (*Below*) Rupture of right leaf of diaphragm. In this patient, right lobe of the liver was displaced into right side of the chest and, on superficial inspection, suggested subdiaphragmatic (subcapsular hepatic) hematoma. Note gastric dilation. This same patient had an "insignificant" fracture of the pubis and rupture of the bladder into the space of Retzius. (*Right*) Excretory pyelogram, Foley catheter in bladder.



liver, the small intestine, the bladder, the pancreas, the colon, the stomach, and the diaphragm. It is noteworthy that, unlike penetrating abdominal wounds in which visceral injuries are commonly multiple, single organs are affected in approximately 80 per cent of cases. However, while infrequent, the dire consequences of missing concomitant injuries make their recognition especially important.

1. *Genitourinary system.* The kidney is the organ most frequently involved by blunt abdominal trauma, especially when the trauma results from direct violence to the flank. Fracture of the lower ribs must raise the suspicion of injury to the kidney and liver on the right side and to the kidney and spleen on the left. Gross hematuria, as observed in a voided specimen of urine or in a specimen obtained by catheter, indicates a wound somewhere along the urinary tract. The intravenous pyelogram and cystogram, along with careful physical examination, are useful for localizing the site of injury.

In the absence of continued bleeding, contusions and lacerations of the kidney are best treated conservatively. Paralytic ileus is often a distressing accompaniment of this lesion, presumably secondary to interference with the extrinsic autonomic innervation of the gastrointestinal tract by retroperitoneal hematoma. Constant nasogastric suction is necessary to maintain de-



Fig. 5. Excretory pyelogram in boy two and a half months after blunt abdominal trauma with rupture of the left kidney. The disruption involved only the lower pole of the kidney. It was possible to resect this damaged portion and to reconstruct remaining part of the kidney without encroaching upon the renal pelvis. Pyelogram is normal except for absence of major inferior calyx of the left kidney. Picture eight minutes after intravenous injection of radio-opaque solution.

compression until gastrointestinal function returns. Operative intervention is indicated when there is evidence of continued hemorrhage into the perinephrium or the paranephrium. Suffice it to say here that, when operation is decided upon, nephrectomy is the usual result. Sometimes, however, it is possible to resect the damaged part of the kidney and preserve the intact portion (figure 5). The preserved part of the kidney must be large enough to include most of the pelvis or at least enough of the pelvis so that closure of it will not end in stricture at or proximal to the ureteropelvic junction.

Contusion of the bladder can be treated by external drainage by an inlying urethral catheter. However, when there is evidence of rupture or laceration of the bladder wall so that its lumen communicates with the peritoneal cavity with the posterior retroperitoneal tissues or with the space of Retzius, the defect in the bladder must be repaired by suture, and the bladder must be placed on constant drainage. Traumatic lesions of the bladder too often go undetected, with catastrophic results.

2. *The spleen.* The spleen is usually injured by blunt direct trauma in the left flank over the

costal margin. Injury should be suspected when the lower ribs on the left side are fractured. Tenderness coupled with spasm in the left upper quadrant with or without fractures of the sixth to twelfth lower left ribs should lead to the diagnosis of laceration of the spleen. The persistence and spread of tenderness and spasm are indications for laparotomy.

A repeat x-ray examination of the abdomen for comparison with the initial film can show changes which may sufficiently increase the suspicion of rupture of the spleen to indicate laparotomy. The second film may show increased haziness in the left upper quadrant of the abdomen, depression of the gas-outlined splenic flexure of the colon, and displacement (figure 6) or serrations of the greater curvature of the gas-containing stomach.

These physical and roentgenologic findings accompanied by an increase in the white blood cell count and a persistently elevated pulse rate despite a maintained blood pressure are sufficient signs to warrant exploration for ruptured spleen.

Contusion of the spleen with the formation of subcapsular hematoma presents a special problem. In such cases, tenderness is often found

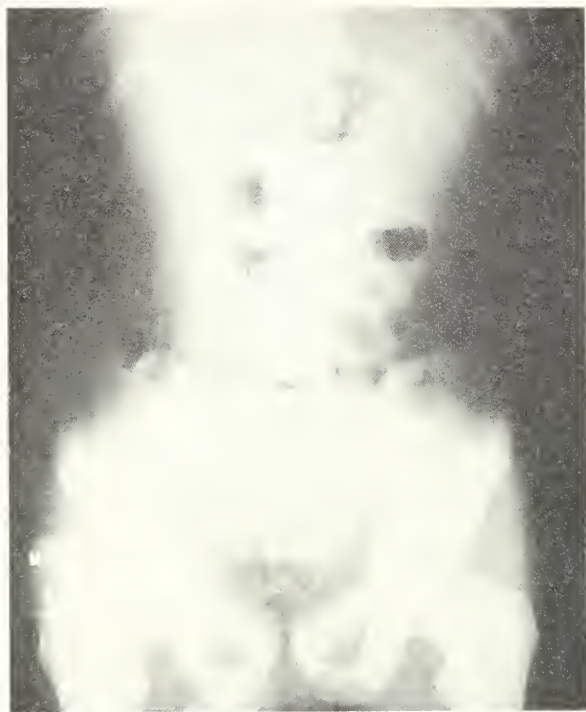


Fig. 6. Plain abdominal roentgenogram (recumbent). Note density in left upper quadrant of the abdomen. Stomach, containing nasogastric tube, is displaced to left; splenic flexure of the colon is displaced downward. Patient proved to have a ruptured spleen. Examination some hours earlier was less obvious.

in the left upper quadrant of the abdomen with only minimal and, sometimes, no spasm. The tenderness may remain without spreading for several days until the question arises regarding the advisability of laparotomy instead of discharge from the hospital. Having passed the immediate period after trauma, the stomach can be outlined by means of small amounts of Gastrografin, and further evidence of abnormalities of the spleen can be obtained by the defects visualized in the gastric wall. A decrease in the hematocrit value may or may not have become apparent.

It has been our policy to perform laparotomy in such patients because of the fear that the lesions might represent a subcapsular splenic hematoma which is liable to rupture and lead to fatal or near fatal hemorrhage as long as two to six or more weeks after the accident.⁵ It would be unrealistic to keep such patients under observation in the hospital for this length of time. We prefer to recommend operation if signs persist beyond thirty-six hours after injury. At operation, lacerations or subcapsular hematomas of the spleen have been found in the majority of such patients. Splenectomy is done without at-

tempting to repair a laceration. It has been suggested that, in infants and children below the age of 2 years, the repair of small lacerations might be considered because of the useful role of the spleen in the control of infection in this age group. The evidence to support this function of the spleen is very doubtful, and, therefore, the spleen with significant laceration should be removed regardless of age. Preservation of the spleen might be considered in only the most trivial laceration or puncture wound.

3. *The liver.* This organ alone is injured in approximately one tenth of patients who have incurred blunt abdominal trauma, and such injury is associated with a high mortality. The injury is seldom seen as a single, sharp linear wound. Commonly, the laceration appears stelliform. It is a well known fact that, even in the face of rather large lacerations of the liver, bleeding has usually stopped by the time laparotomy is done. Nevertheless, operation is indicated even when this is believed to be the only intra-abdominal lesion. The edges of the wound can be loosely sutured, and the area of drainage can be exteriorized by means of one or more appropriate drains. The danger of bile peritonitis is thus avoided. Occasionally, fragments of traumatically disrupted liver reach the lungs as pulmonary emboli,⁶ much as fragments of skeletal muscle do. The clinical significance of this needs to be assessed.

Avulsion of the gallbladder is not uncommon in association with blunt wounds of the liver. The gallbladder is removed down to the common duct when this can be done safely. If the hematoma, which may develop as the result of the injury, makes visualization of the common duct and adjoining structures hazardous, it is preferable to remove the contused or devitalized portion of the gallbladder and to drain the organ as a cholecystostomy.

4. *The gastrointestinal tract.* Lesions of the intestine and its mesenteries are fairly common after blunt abdominal trauma but not as common as injuries of the solid organs. Contusion of the bowel wall or mesentery can, at best, be only suspected, but rupture of the stomach, duodenum, or the large intestine into the free peritoneal cavity results in readily detectable, often dramatic, pneumoperitoneum and peritonitis. Rupture of the small intestine, however, between the ligament of Treitz and the ileocecal valve characteristically presents no evidence of pneumoperitoneum either by roentgenogram or by physical examination (figure 3). The physical findings are those of localized, spreading, or generalized peritonitis.

Similar signs of peritonitis sufficient to warrant laparotomy are found with contusions of the mesentery and of the wall of the large and small intestine. Such lesions can produce signs of localized and of spreading peritonitis. Since it is not possible to distinguish such injuries from rupture of the small intestine or from lacerations of the liver and spleen, the physical findings make laparotomy mandatory.

Especially treacherous are the cases of patients in whom blunt abdominal trauma has resulted in retroperitoneal rupture of the intestine. This kind of damage occurs in those parts of the colon which lack a mesentery, in the terminal ileum, in the first portion of the jejunum, and in the duodenum. These areas must be inspected at the time of laparotomy, and, if there is gross evidence of retroperitoneal contusion or infiltration, it is well to mobilize that part of the intestine in order to inspect the retroperitoneal portion of the viscus. Rupture of the viscus, however small, can be detected by sponging the area with a dry, white gauze. The stain from intestinal contents will usually be apparent on the gauze. These perforations should be carefully repaired and the area exteriorized by means of an appropriate drain sutured with plain catgut to the retroperitoneal tissue adjacent to the bowel at its site of perforation. Conservative management of such lesions is ill advised.

In two patients whom we have recently seen, retroperitoneal rupture of the duodenum, second and third portions, respectively, was suspected preoperatively on the basis of the roentgenologic appearance.^{7,8} There was partial obliteration of the psoas shadow by density and bubbly gas, and there was moderate lumbar scoliosis with convexity toward the left (figure 7). Failure to consider seriously the possibility of this diagnosis in casualties with blunt abdominal trauma usually results in making the diagnosis at autopsy.

5. *The pancreas.* Because of its position across the spinal column, the pancreas may be injured by blunt violence to the abdomen, such as in the steering-wheel trauma of automobile accidents. Minor degrees of such pancreatic injuries usually go unrecognized or result in varying degrees of pancreatitis. Mathewson and Halter⁹ report that trauma is an important etiologic factor in pancreatitis.

Actual disruption of the pancreas is less common. Its signs and symptoms are indistinguishable from those of acute peritonitis from other traumatic causes, with the exception of that secondary to rupture of the stomach or colon in which pneumoperitoneum is usually detectable both clinically and by roentgenogram. Lacerations

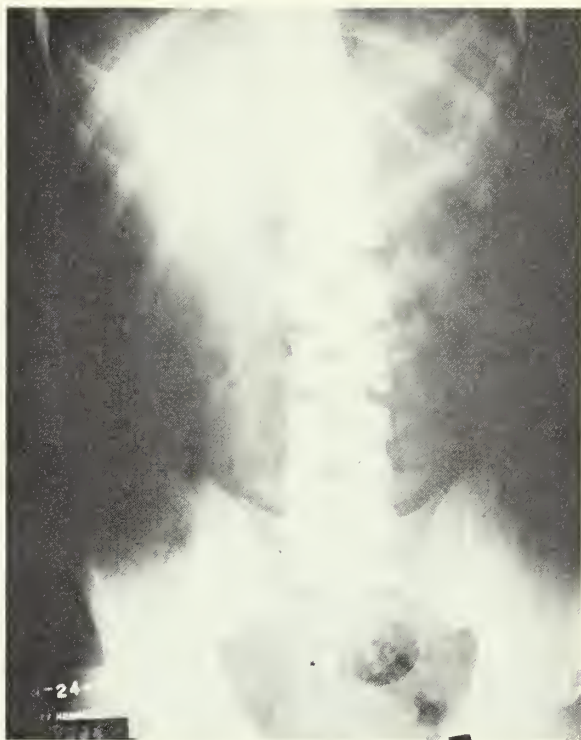


Fig. 7. Plain roentgenogram of young man with retroperitoneal rupture of second portion of the duodenum following blunt abdominal trauma in a bicycle accident. Note lumbar scoliosis with convexity to right. Right psoas margin is rendered indistinct in its lower half by infiltration with "bubbly" gas shadows.

of the body and tail of the pancreas may affect only the lesser peritoneal sac. In such cases, the usual difficulties are encountered, as with the diagnosis of peritonitis from other causes in the lesser peritoneal space. When the nature of the injury leads the surgeon to suspect possible injury to the pancreas, he should inspect the body and tail of the pancreas through an opening in the gastrocolic ligament.

Fragments of the tail or body of the pancreas to the left of the superior mesenteric vessels are best resected. When this is not feasible, a drain is sutured to the traumatized area in the hope of exteriorizing it and coping with the pancreatic fistula later if one does form. There is disagreement with regard to the use of drains in injuries of the pancreas. Our policy is to make use of them whenever the pancreas is wounded to any significant extent. When they are used, however, it is important to affix them to the point they are intended to exteriorize. A carefully placed, readily absorbable suture is used for the purpose. This exteriorization by means of a drain must be accomplished through a direct route and not a circuitous one.

The formation of pseudocysts of the pancreas is common after blunt abdominal trauma. The pancreatic injury may not have been detected at the time of the accident, which may have occurred a few months to several years before the diagnosis of pancreatic cyst. The treatment of these cysts is preferably by internal drainage, but, as in pseudocysts from other causes, marsupialization is sometimes necessary.

6. *The diaphragm.* The diaphragm is not commonly injured by blunt abdominal trauma. It may be lacerated by the ends of fractured ribs, and the injury should be suspected when fracture of the lower ribs on either side is detected clinically or by roentgenogram. Occasionally, however, the diaphragm ruptures as the result of violent pressure from the abdominal side. The wound may be complete, passing through both the pleural and peritoneal visceral layers, or incomplete, when one or more of the diaphragmatic layers remain intact. It is noteworthy that the majority of patients with eventration of the diaphragm have a history consistent with antecedent traumatic diaphragmatic rupture.

Injury to the diaphragm from nonpenetrating abdominal wounds is usually found on the left side, for here there is less protection from the liver than there is on the right side. The diagnosis is easier when the lesion occurs on the left because gas-filled organs, such as stomach, colon, or small intestine, are readily detected in the left side of the chest by examination or roentgenogram (figure 4, *left*). On the right side, herniation of hollow organs is less common, and the injury is easily mistaken (figure 4, *center*) for elevation of the diaphragm because of a large subcapsular hematoma. However, gastric dilation is noted, a useful sign of rupture of the diaphragm with dislocation of the right lobe of the liver into the chest and duodenal torsion and obstruction.

Lacerations of the diaphragm should be repaired without delay as soon as the diagnosis is made. A thoracoabdominal exposure is preferred because it permits ready access to the pleural surface of the diaphragm for a meticulous two-layered repair with nonabsorbable sutures and because it permits careful exploration of the abdomen for concomitant injury. In contradiction to the general principle of the important role of rest in wound healing, the phrenic nerve is not blocked either permanently or temporarily. Intercoastal tube water-seal drainage is employed for twenty-four to forty-eight hours, when the tube is withdrawn unless there is evidence of infection. Abdominal lesions requiring drainage, such as lacerations of the liver, are separately drained through the abdominal wall.

Other injuries. Unlike blood vessels in the periphery, lesions of the major arteries and veins of the abdomen rarely occur after nonpenetrating trauma. Hematomas, sometimes massive, do form retroperitoneally and in the several mesenteries, but we have not encountered a lesion of the vena cava, of the aorta, or of their major branches after blunt abdominal trauma.

Perforations of the stomach, small intestine, and colon are not as common after blunt trauma as they are after penetrating wounds of the abdomen. The principles of management are similar in both types of wounds. Perforations of the stomach and small intestine are simply repaired. When they are multiple, it may be more simple to resect the involved portion of intestine than to repair a great many openings in a short loop of intestine. In the case of lesions of the colon, we believe that it is safest to repair these lacerations as perforations of the small intestine are repaired but, in addition, to provide proximal defunctioning or, at least, decompression. This policy has been considered ultraconservative, and, in fact, in many cases, it has probably been unnecessary. However, in cases we have seen in which proximal defunctioning colostomies might have averted trouble, the price paid was high.

COMMENT

It is apparent that the surgeon is confronted by very special problems when dealing with blunt abdominal trauma. Of first importance is the recognition of serious internal injury in the absence of visible external signs. We have emphasized the fact that the layman cannot be expected to exercise sophisticated judgement in deciding whether he or his child is becoming better or worse. It is safest to follow a conservative policy and to admit to the hospital for observation those individuals who fulfill a few simple criteria which have been outlined. To be sure, such a conservative policy has unnecessarily used hospital beds in some instances, but, in recent months, our patients have not died on the way to or from their homes or the local jail.

The next question concerns the decision for operation, and this applies not to the obvious case in which decision presents no problem but to the patient who is hospitalized for observation. Operative intervention is indicated by the development of certain signs, among which the most important are the following:

1. Increasing signs of irritation of the parietal peritoneum—tenderness and spasm.
2. Accumulation of intraperitoneal fluid—shifting abdominal dullness or referred pain of subdiaphragmatic origin.

3. Adynamic ileus—intra-abdominal hemorrhage, peritonitis from rupture of a hollow organ, or retroperitoneal bleeding.

4. Cardiorespiratory signs and symptoms—rupture of diaphragm with herniation.

5. Fever and roentgenologic findings of retroperitoneal rupture of duodenum or other nonperitonealized parts of the intestine.

6. Roentgenologic signs of increasing size of localized mass—laceration or subcapsular hematoma of spleen or liver.

7. Development and progression of early clinical signs of shock.

It is well to emphasize that, in this group of patients, shock is *nearly always* attributable to oligemia. In most cases, whole blood has been lost, and progressive hemodilution is detectable until the deficit has been corrected by transfusion. In some patients, there is a combination of whole blood and plasma loss. Depending upon the relative importance of the two, the hematocrit value may be normal or even high. Oligemia nevertheless exists and must be corrected. Fat embolism and pulmonary and mediastinal embarrassment from rupture of the diaphragm and herniation are uncommon but should be kept in mind as interfering with the management of shock.

Our experience during the past twelve months may be cited as illustrative of this policy, the principles of which are cited in this paper. During this period of time, 6,578 patients who had incurred some form of trauma were seen in the accident room. From this number, 264, or 4 per cent, were admitted to the hospital. Sixteen had experienced blunt abdominal trauma. Ten were kept under observation and discharged when it had become clear that there was no intra-abdominal injury. Six of the 16 were operated upon. Only 1 of these proved to have no important intra-abdominal injury. The other 5 had serious lesions of the spleen, kidney, mesentery, and other structures. No death occurred among this group of patients with blunt abdominal trauma.

Brief mention has been made of the special problems presented by injury to the various abdominal organs in this kind of trauma. The grave error of missing a retroperitoneal rupture of the duodenum has been emphasized, and the importance of external drainage for hepatic wounds has been noted. Lesions of the stomach, duodenum, and small intestine are managed differently from those of the colon. Defunctioning proximal colostomy is an excellent safety measure in the latter.

Some question may arise concerning our policy with regard to the patient who, after two days of observation, still presents signs and symptoms of peritoneal irritation in the left upper quadrant of the abdomen and in whom a small rupture of the spleen, subcapsular splenic hematoma, or other lesion cannot be ruled out with certainty. We believe that it is safer to operate upon such patients than to discharge them or to observe them for much longer periods of time. It is well known that subcapsular hematomas may rupture many days or weeks after injury. Therefore, observation would have to be very prolonged—at least two weeks⁵ and preferably much longer. Upon exploration of the abdomen, splenectomy is done even for very small lacerations or hematomas of the spleen. Only the very slightest lacerations or puncture wounds are repaired when no more than a single mattress suture is required. The spleen is notoriously difficult to repair.

CONCLUSION

From the clinical standpoint, two important aspects of the management of patients who have incurred blunt abdominal trauma deserve emphasis. The first is the recognition of the patient who must be closely observed for twenty-four to forty-eight hours before one can conclude that he has no significant intra-abdominal injury. The second is the recognition of the need for operative intervention, even without precise diagnosis of the specific injury, before irreparable damage results from bleeding and infection.

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Diagnosis and Treatment of Cardiac Emergencies

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RATIONAL and successful treatment of cardiac emergencies requires a number of things. It requires the maximal use of the facilities always present: one's eyes, ears, and hands. It requires that the recognition of these emergencies be carefully thought out beforehand. It requires that the physician be constantly critical of his own diagnoses in the light of changes in the clinical picture and response to therapy.

ACUTE PULMONARY EDEMA

One practical example of these precepts is in the management of acute pulmonary edema. The majority of such cases will be on the basis of low-output failure; examples of this, in adults, are coronary artery disease, hypertension, rheumatic valvular disease, and advanced renal disease. The urgent treatment of this condition must of necessity be individualized. In outline, however, it generally consists of use of morphine in adequate doses (10 to 15 mg. intravenously in the average-sized adult); oxygen by mask, preferably with 50 per cent alcohol in the nebulizing solution; rotating tourniquets (to occlude venous but not arterial flow) and/or phlebotomy (with letting of 500 to 700 cc. of blood); mercurial diuretics by injection; and digitalis. The form of the latter that I prefer to use is Cedilanid, 0.8 mg. intravenously initially, followed by 0.4 mg. intravenously every two hours to a total of 1.6 to 2.0 mg., depending on the size of the patient and the therapeutic response. If the dosage of Cedilanid is adjusted in this way, overdigitalization is less likely to occur; since the response to the initial measures is usually prompt, the patient's condition is not compromised by the delay in full digitalization.

To treat all such patients the same, however, would be disastrous. The key to successful management is continuing observation of the patient, modifying the above outline of therapy on the basis of individual circumstances. The emergency is never so great that one cannot do a rapid but incisive physical examination and attempt to glean pertinent historical data. Consideration must be given to prior digitalization, coexistent renal disease, and other complicating factors. The distinctive sensitivity of the patient with an acute myocardial infarction to digitalis intoxication must be recognized. If acute pulmonary edema is on the basis of marked hypertension, cautious adjustment of dosage with ganglionic blocking agents may be lifesaving, but care must be taken not to compromise cerebral, coronary, and renal blood flow by overly energetic treatment.

Certain discrepancies in the usual picture of acute pulmonary edema, such as the relative bradycardia of the myxedematous patient or the disproportionate extent of cardiac dullness beyond the point of cardiac impulse in pericardial effusion, are, if recognized, crucial clues to accurate diagnosis and subsequent therapy. Finally, pulmonary edema on the basis of high-output failure is seen infrequently enough that it is necessary to be reminded of it often; probably every physician has had the unhappy experience of being so engrossed in the acute pulmonary edema as to completely overlook the underlying disease on which it is based. Examples of high-output failure include thyrotoxicosis, which, especially in the aged, may be far from obvious; severe anemia such as pernicious anemia in relapse; thiamine deficiency; and arteriovenous aneurysms. In these conditions, use of digitalis will either be of little value or actually deleterious, and treatment should obviously be directed at the primary disease.

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One practical point which seems to consistently escape inclusion in the standard textbooks is the amount of digitalis to give after the emergency treatment phase. If Cedilanid has been used intravenously, approximately one-half of the digitalizing dose will be excreted in the ensuing twenty-four hours, providing that kidney function is adequate. The dosage of digitalis for the same twenty-four hours, then, will be one-half of the digitalizing dose of a slower acting preparation: usually 0.8 gm. of digitalis folium or 0.8 mg. of digitoxin, in divided doses. On the following day, approximately one-fourth of the digitalizing dose will be required. Here again, obviously, the clinical response of the patient takes precedence over any such dosage schedules, which serve only as a general guide.

ACUTE MYOCARDIAL INFARCTION

At present, direct attack on acute coronary thrombosis, such as the use of intravenously given fibrinolytic agents, is still an experimental procedure. Therefore, treatment is directed at preventing complications of the myocardial infarction and achieving optimal healing of the infarcted area. Narcotics in adequate doses are used intravenously or intramuscularly to relieve pain and ensure sedation. Oxygen by mask may be used if respiratory distress is present or if it appears to make the patient more comfortable. The three acute complications which commonly occur at the time of infarction are shock, pulmonary edema, and the arrhythmias. Levophed remains the most potent hypertensive agent generally available, but it must be given intravenously and the rate of infusion continually adjusted in relation to the minimum blood pressure necessary to maintain adequate circulation. Urine output and specific gravity are one practical measure of this. For milder degrees of shock, Aramine, 10 mg., may be used subcutaneously or intravenously but should be abandoned in favor of Levophed if the response is not prompt. Unfortunately, acute myocardial infarction complicated by severe shock presents an extremely poor prognosis even with the most prompt and careful therapy.

The management of pulmonary edema has been outlined above, but digitalis must be used with special caution, due to the enhanced irritability of the ischemic myocardium. If digitalis is required at all, it should probably be given concurrently with Pronestyl or quinidine (unless complete heart block is present) in an attempt to prevent paroxysmal tachycardias. The specific management of arrhythmias will be discussed below.

Late complications of myocardial infarction include phlebothrombosis, pulmonary embolization, and propagation of the coronary thrombosis, for which anticoagulation at two and one-half to three times the control is prophylactic, and ventricular aneurysm and/or rupture, which one hopes to prevent by absolute bed rest. Use of atropine in the initial phase of an acute infarction is somewhat controversial, but experimental evidence would indicate a beneficial effect on coronary blood flow if it is given very early in the course. Atropine should not be given if tachycardia is present.

COMPLETE HEART BLOCK

The clinical course of complete heart block is quite variable: it may become evident as severe circulatory embarrassment at the onset or it may be present for many years without symptoms. In complete heart block, the cardiac pacemaker is located in one or more sites in the ventricle and characteristically drives the heart at the inherent ventricular rate of 30 to 50 beats per minute. The rhythm is regular, and the first sound at the apex is typically split, due to ventricular asynchrony, and variable in intensity, due to the atrioventricular (AV) dissociation. A compensatory systolic hypertension is usually present. Coronary and cerebral circulation are always precarious, and at any time asystole may occur—the classical Adams-Stokes syndrome—resulting in syncope, convulsions, and, if not corrected spontaneously or therapeutically, sudden death. A similar sequence may also be caused by paroxysmal ventricular tachycardia complicating complete heart block. In simple asystole, stimulation of the myocardium to reestablish a pacemaker may be accomplished, depending on the circumstances, by pounding on the chest, by inserting a needle into the myocardium, or by applying electrical stimulation to the chest wall. The latter is the treatment of choice. The most direct approach, manual massage of the heart, should be considered only when there is immediate access to an operating room and a well-trained surgical and anesthetic team is on hand. Molar sodium lactate intravenously, which acts by increasing the reactivity of the basic ventricular pacemaker and by improving the AV conduction, is probably the best treatment for paroxysmal ventricular tachycardia complicating complete heart block. This drug and Isuprel are of greatest use between attacks in preventing episodes of asystole or tachycardia. Pronestyl and quinidine are strongly contraindicated in any case of AV dissociation, since they may abolish the ventricular pacemaker and produce

cardiac arrest. Paroxysmal ventricular fibrillation is rarely diagnosed (outside the operating room) and is almost invariably fatal. Cardiac massage and defibrillating current are the only treatments available.

Fundamentally, the ideal management of complete heart block lies in an attempt to reestablish normal AV conduction and thereby prevent Adams-Stokes episodes. If the heart block is acute, this may be accomplished by giving atropine, which abolishes the vagal depression of the AV node; by giving aminophylline, which may improve nodal circulation; or, in the case of acute myocardial infarction adjacent to the AV node, by giving adrenal steroids, which may reduce the zone of inflammation involving the node so as to allow normal conduction. Short of this, interim treatment consists of use of drugs such as Isuprel or of monitored electrical stimulation.

PAROXYSMAL TACHYCARDIAS

The commoner tachycardias can usually be differentiated at the bedside. This is a point of considerable practical importance, as well as a challenge. Attention to the rate, rhythm, heart sounds, and response to vagal stimulation will usually provide the correct diagnosis. Electrocardiographic confirmation is, of course, highly desirable but is not always available.

Paroxysmal supraventricular tachycardia (the pacemaker being situated in an ectopic atrial focus or in the node) is probably the commonest form. It may occur in otherwise normal hearts and may be fairly well tolerated initially, or, when superimposed on a diseased heart, it may precipitate acute failure or infarction. It is characterized by an absolutely regular rate of 150 to 250 beats per minute which frequently will subside spontaneously or respond to such simple procedures on the part of the patient as the Valsalva maneuver. The heart sounds are normal in timing and intensity.

Carotid sinus massage will, if effective, cause an abrupt cessation of the tachycardia, followed by a variable period of asystole (usually a few seconds). It is because of the asystole that special care must be taken in this maneuver and carotid sinus massage stopped as soon as auscultation over the precordium reveals any change in rate. In the aged, carotid massage should be done quite gently, to avoid interference with cerebral circulation. After the period of asystole, a sinus rhythm may be restored or the tachycardia may resume, but the dramatic response is diagnostic. Should vagal stimulation fail to control the tachycardia, and if the situation is

not urgent, sedation with sodium luminal, 2 gr. intramuscularly, may be tried. Where prompt action is necessary, however, the drug of choice is Cedilanid, 1.2 to 2.0 mg. intravenously, depending on the size of the patient, in divided doses. Repeated carotid sinus massage when the patient is partially or fully digitalized is frequently effective.

Finally, if the tachycardia still persists, quinidine should be added next, either 6 gr. orally every three hours or, if the urgency of the situation warrants it, as an intravenous drip of 10 gr. in 100 cc. of 5 per cent glucose, with electrocardiographic monitoring. Other drugs which have been recommended in treating paroxysmal atrial tachycardia are parasympathomimetic agents such as Prostigmin or Tensilon, which mimic the vagal effect on the atrioventricular node, and pressor amines such as Neosynephrine and Aramine, which, by their hypertensive effect, result in an "internal" carotid sinus stimulation.

The cardiac rate in atrial flutter will depend on the number of atrial impulses which are able to activate the ventricles. In most cases, only one-quarter to one-half of the impulses pass the AV node, resulting, therefore, in a 4:1 or 2:1 block. Conduction of 1:1 does occur but is quite rare. In a typical case of flutter causing tachycardia, the apical rate will be 120 to 175, with 2:1 block of the flutter rate of 240 to 350. The degree of block is usually constant at least part of the time, so that a basic regularity can be detected. The heart sounds are normal in timing and intensity. Of greatest value to the experienced observer is the ability to detect and count atrial pulsations in the neck veins, making the diagnosis certain. Carotid sinus pressure may increase the degree of AV block, causing a sharp drop in rate (usually to one-half the initial rate), but the tachycardia quickly recurs, despite continued stimulation. Again, this specific response, if present, is highly diagnostic, even though not curative. Standard treatment consists of full digitalization to slow the ventricular rate, followed, if necessary, by quinidine to convert the flutter to sinus rhythm.

As in atrial flutter, in atrial fibrillation the apical rate is governed by the degree of block which the AV node presents to the chaotic atrial activity of some 400 impulses per minute. The rhythm is totally irregular, and a marked discrepancy between the apical and radial rates is noted, especially at the faster rates. Carotid pressure usually produces a gradual but unimpressive and transient slowing of the rate. Full digitalization is employed to control the tachycardia.

Quinidine should not be used until the ventricular rate is below 90 beats per minute, since conversion to atrial flutter with a very rapid ventricular response may otherwise occur.

Paroxysmal ventricular tachycardia is the most serious disorder of this group, since its natural progression is to ventricular fibrillation and death. It occurs almost invariably in severely diseased hearts or as a manifestation of digitalis intoxication. At a rate of 150 to 220 beats per minute, the rhythm may appear superficially to be regular, but comparison of minute-to-minute counts will reveal definite irregularity. The first heart sound at the apex is widely split, due to ventricular asynchrony, and may be variable in intensity, due to independent atrial activity. Carotid sinus pressure has no effect. Pronestyl, which is safer and more effective than quinidine, should always be on hand for treating this emergency. The dosage is 100 mg. (1 cc.) intravenously every minute until the arrhythmia is abolished. The practical limitation on dosage is 1,000

mg., but a higher dosage may be required in a few critical cases. Electrocardiographic monitoring to detect significant lengthening of the QRS interval is a very important guide to toxicity. When frequent premature ventricular contractions occur in the course of an acute myocardial infarction, administration of Pronestyl, 250 mg. every six hours orally or intramuscularly, should be started immediately, unless the specific contraindication of AV dissociation is present.

Finally, it cannot be stressed too often that digitalis can and does produce virtually any arrhythmia. Unpredictable factors such as individual sensitivity to digitalis and the accentuation of digitalis toxicity by hypokalemia (due to abnormal fluid loss, diuresis, chlorothiazide administration, etc.) make digitalis a somewhat less than obvious factor in many arrhythmias. When digitalis toxicity is suspected, use of the drug should be stopped at once and, unless contraindicated, potassium salts given orally or intravenously.

THE CHOICE of patients under 20 years of age for surgery for rheumatic heart disease should be based upon the same criteria used for older subjects. Surgery should be performed when a child has an anatomic defect correctible at reasonable risk and no persistent rheumatic activity. Antibiotics should be administered postoperatively in doses ensuring protection from bacterial infection. In children with rheumatic disease, serious cardiac manifestations such as arrhythmias, cardiac enlargement, and congestive failure are usually attributed to myocarditis rather than valvular defect. However, symptoms of heart failure may be due to a valvular lesion. Surgery is well tolerated when a mechanical impediment is the sole cause of clinical deterioration of a child. If proper precautions are taken, valvular surgery will not reactivate rheumatic disease in young patients.

A. N. BREST, J. URICCHIO, and W. LIKOFF: Valvular surgery in the young patient with rheumatic heart disease. *J.A.M.A.* 171:249-251, 1959.

The Management of Congestive Heart Failure

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MANY EXCELLENT REVIEWS have covered the subject of the management of congestive heart failure completely, yet we often see patients who, despite an ever-increasing number of new drugs, suffer from recurring or chronic symptoms of "intractable heart failure." Except for those with terminal myocardial insufficiency, there are very few patients whose heart failure is nonresponsive to therapy. The degree of success in an individual case is determined by the diligence with which the well-known principles of treatment are applied. This discussion will concentrate on some details which often determine the success of treatment.

Regardless of its etiology, when congestive heart failure occurs, impaired cardiac function causes a series of pathophysiologic changes which result in renal retention of sodium and water, increased blood volume, and alteration of the water and electrolyte content of tissue and body spaces. The symptoms which develop are determined by these changes, and treatment is aimed at their correction. The ultimate goal of therapy is to improve cardiac function and to prevent retention of excess sodium and water and thus to return the patient to as normal and symptom-free a life as possible.

The usual symptoms of heart failure—dyspnea, orthopnea, and edema—are well known and usually readily recognized by the patient and the doctor. But the patient who is well instructed and closely watched can usually avoid development of these symptoms. The daily weight, determined on arising, is a reliable measure of change in fluid content of the body and also permits evaluation of the effect of therapy. Patients soon learn the significance of increasing weight and can regulate their medications, rest, diet, or visits to the doctor before symptoms develop. Insomnia that occurs because "I get restless in bed, feel better sitting" or an unexplained cough, particularly if it occurs when lying down, may be signs of impending left heart failure. The appearance of symptoms or signs of cardiac insufficiency is an indication for a reappraisal of the

patient and his treatment. Two questions should be asked:

1. Has an increased load been imposed on the circulation or has something happened to decrease the efficiency of the heart? Failure may be precipitated by myocardial infarction, rheumatic activity, progressing valvular deformity, pulmonary embolism, or arrhythmias. Appropriate treatment of any of the foregoing by steroids, cardiac surgery, anticoagulants, or quinidine may permit otherwise intractable heart failure to be controlled. Other disease states, such as fever, hyperthyroidism, hypothyroidism, hypertension, arteriovenous aneurysms, or anemia, could be contributing factors and if present should be corrected. Oftentimes, increased exertion or anxiety associated with change of work, residence, or manner of life may be responsible. For the housewife, guests, social activities, or growing children may be found to be the added load which led to failure. Only when all conditions contributing to the inefficiency of the heart, or unduly increasing the cardiac load, are treated as adequately as possible can maximal recovery from heart failure be obtained.

2. Are the various means of treatment being used to their maximum? Rest, digitalis, diuretics, and diet, the principal tools for the treatment of heart failure, are well known and used by all physicians. In many patients, heart failure will respond rapidly to one or a combination of these; but, in the problem case, the results will be determined by the care and precision with which all phases of the treatment are applied.

REST

The importance of rest has been recognized from earliest times. To be effective it should be mental as well as physical and should be as complete as needed to control heart failure. For the ambulant patient it may suffice to reduce his working hours, prescribe rest periods during the day, and assure restful sleep. When a patient is not responding to therapy, bed rest, preferably in a hospital, until signs of failure have cleared can usually be followed by a gradual return to a level of activity that was previously impossible. Many patients have found that diuretics are more effective if combined with bed rest,

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and, unless they are bothered by the interruption of sleep, they will prefer to take them at bedtime because of the better diuresis obtained.

DIGITALIS

The value of digitalis is dependent upon its ability to increase the output of the heart in failure and to slow the ventricular rate, especially in cases of rapid auricular fibrillation. The prime indication for digitalis therapy is congestive heart failure resulting from defective myocardial contraction, from the inefficiency of excessive ventricular rate, or from a combination of the two. To have maximal effect, digitalis must be given in adequate dosage. Many attempts have been made to determine the correct dosage of the various preparations, but the original instructions of William Withering remain the best: "Let the medicine therefore be given . . . until it either acts on the kidneys, the stomach, the pulse, or the bowels." That is, until there is diuresis or slowing of the pulse or toxic signs of nausea, vomiting, or diarrhea.

If the patient has had no digitalis for the previous two weeks, and if speed is urgent, as in rapid auricular fibrillation with failure, digitalization may be accomplished by the intravenous route with ouabain, 0.5 mg.; digoxin, 1.0 mg.; or lanatoside C, 1.6 mg. (If indicated clinically, the initial dosage may be followed at two-hour intervals by additional injections in one-fourth of the initial dosage, but extreme caution must be exercised to avoid overdosage.) In most cases, and when the state of digitalization is unknown, the oral route is preferred. The physician should use the form of digitalis with which he is most familiar, for the results are determined more by the manner of usage than by the particular brand used. I prefer a shorter-acting glucoside (Digoxin) which may be given in repeated doses every four hours until a desired therapeutic effect is obtained or until toxicity results. In this latter situation, discontinuance of the short-acting, rapidly excreted drug permits a rapid clearing of the toxic symptoms. After digitalization is accomplished, a maintenance dose of digitalis is continued. Occasionally nausea may be caused by the heart failure, and so uncertainty arises as to the further use of digitalis. If there is any question in this regard, an electrocardiograph may be checked for digitalis effect.

DIET

In patients with congestive failure, the kidneys do not excrete the amount of sodium in the aver-

age diet, usually 3 to 6 gm. a day. This amount, if retained entirely, will hold in the body from a pint to a quart of water to maintain osmotic equilibrium. The amount of dietary sodium should be reduced to a level which permits the patient to remain edema-free. Usually a diet containing 1 to 2 gm. of sodium affords sufficient restriction; if the desired result is not obtained, greater care should be exerted and all possible sources of sodium eliminated. Often overlooked are such sources as sodium-containing medications, seasonings, or foods with high natural sodium content. The manual, "Food For Your Heart," available from the local or the American Heart Association, is an excellent guide and help to the physician and the patient. It is important that the diet, though low in sodium, remain palatable, and patients should be checked carefully to be sure that their nutritional needs are being met. If the patient is overweight, it must be remembered that a reduction in cardiac work follows reduction in weight, and a low-calorie diet should be prescribed.

The question of fat content of the diet is still undecided, but, except in the presence of hyperlipemia, there is no conclusive evidence that the fat content of the diet should be altered other than to attain a suitable caloric level. William Withering again has given practical advice, this time regarding fluid intake: ". . . the patients should be enjoined to drink very freely—I mean they should drink whatever they prefer, and in as great quantity as their appetite for drink demands. This direction is the more necessary, as they are very generally prepossessed with an idea of drying up a dropsy, by abstinence from liquids and fear to add to the disease by indulging their inclination to drink."

DIURETICS

Diuretics, and particularly the parenterally given mercurials, have become the most important and effective part of the treatment of congestive heart failure. Preparations for subcutaneous injection such as mercaptomerin (Thiomerin) sodium, meralluride (Mercurhydrin) sodium, and merthoxylline (Dicurin) procaine permit administration not only by the physician or nurse but by the patient or some member of his family. The dosage may vary from 0.2 cc. to 2 cc., with an average dose of 1 cc. The large diuresis with a weight loss of 5 to 15 lb. which may follow a single injection is dramatic but is needlessly prostrating to the patient. With daily weights used as a guide, an attempt should be made to produce a diuresis of 1 to 4 lb. a day. After an initial injection, diuresis may continue for sev-

eral days, but when it ceases the injection should be repeated.

Sensitivity to the mercurials may be manifested by local or systemic reactions and makes it unwise to use them further for that patient. It is also felt that mercurial diuretics should not be used in the presence of acute renal disease.

Aminophylline, in dosage of 0.25 to 0.5 gm. intravenously (given over 10 minutes) or rectally in suppository form, or as a powder dissolved in $\frac{1}{2}$ oz. of water and injected with a small bulb syringe, has some diuretic effect and is particularly helpful in increasing the response to injected mercurials.

Ammonium chloride, given as enteric-coated tablets in divided doses of 4 to 6 gm. daily, or acetazolamide (Diamox), 0.25 to 1 gm. daily, if given for three to four days a week will increase the response to injected mercurials.

Chlorothiazide (Diuril, 0.250 to 1 gm. once or twice daily) and hydrochlorothiazide (Hydro-diuril; Esidrix, 0.025 to 0.1 gm. once or twice daily) have proved to be the most effective of the orally given diuretics. Many patients may be maintained in an edema-free state with these drugs alone; others may require many fewer mercurial injections. These drugs increase the excretion of potassium and, if used in larger doses, especially if the patient is eating poorly, may cause hypokalemia. This may be prevented by administration of potassium chloride as 1 gm. 4 or 5 times daily.

Whatever diuretics are used, the patient should be kept free of excess fluid. This can be well shown by the daily weight record. The medication should be given in such form and dosage that the weight remains constant at the edema-free level. It is unfortunate for patients who suffer the symptoms of increasing heart failure to be relieved at weekly or biweekly intervals by an injection of mercurial that leaves them prostrated from the large diuresis. It is much better, if oral medication does not suffice, to have the patient receive frequent small injections of mercurial, using his weight as a guide, much as a diabetic takes insulin depending on the urine test.

Recently the adrenal steroids have proved helpful in patients with resistant fluid retention. They should not be used routinely, but, in the patient under close observation who has not responded adequately to the regimen of rest, digitalis, and diuretics, the adrenal steroids, particularly the newer preparations with minimal sodium-retaining action, may promote a good diuresis when given in usual dosage over several days.

The advent of potent diuretic and saluretic agents introduces new hazards into the therapy of congestive heart failure. A patient undergoing intensive diuretic therapy, particularly if he responds poorly, is in danger of developing electrolyte imbalance. Thirst, weakness, lethargy, muscle pain, hypotension, oliguria, nausea, vomiting, tachycardia, confusion, or coma may be signs of the low-salt syndrome, hypokalemia, acidosis, or alkalosis. These complications may be decreased if rapid diuresis is avoided and the patient takes a normal diet. One of the advantages of the potent diuretics is that they permit the use of more salt, thereby adding to the palatability of the diet and the nutrition of the patient.

Whenever a patient is doing poorly, the blood electrolyte values should be determined and, if they are out of balance, every attempt should be made to correct them. If electrolyte studies are not available, it is often wise to discontinue medication and allow a normal diet for a few days to see if improvement occurs. Then a new start can be made on therapy.

The following case report will illustrate how some of these principles may be applied in practice.

CASE REPORT

A 56-year-old laborer, in November 1957, noted gradually increasing dyspnea, orthopnea, and abdominal bloating. In April 1958 he was forced to stop work and consult his physician. After three weeks of rest and medication at home he had improved sufficiently to return to work. After only two days he was "just as bad as before." For the next six months he continued under treatment but was able to work for only a few days at a time because of recurring congestive heart failure.

In November 1958 this patient was referred for evaluation. The general physical examination was not significant except for signs of congestive heart failure. Blood counts were normal. The electrocardiogram showed left bundle-branch block, and the chest x-ray showed a diffusely enlarged heart with a cardiothoracic ratio of 19.5:33 cm. It was apparent from reviewing his poor response to previous treatment that congestive failure had not been adequately controlled by ambulatory therapy. To initiate treatment he was hospitalized for three weeks. During this period he was digitalized and given mercurial diuretics, aminophylline, Diuril, and a 1-gm. sodium diet. At first he was kept at bed rest in an orthopneic position, but after substantial improvement he was started on a prescribed program of slowly increasing activity. During this period a steady diuresis brought his weight from 198 to 165 lb. At this point it stabilized, but the addition of an adrenal steroid (Decadron, 0.75 mg. three times a day for five days) was followed by a further diuresis to 156 lb. and complete absence of any symptoms of congestive heart failure. After discharge from the hospital he continued on a program of gradually increasing exercise. He followed moderate salt restriction in diet and continued digitalis therapy (Digoxin, 0.25 mg. daily). He has been seen at regular intervals in the office and careful search has been made for any

signs of returning failure. Injections of mercurial (Thio-merin, 1 cc.) were given at intervals as a test for any occult edema. With this schedule, he has remained entirely free of symptoms and has returned to his former work, which he now does without any difficulty. The heart diameter has decreased from 19.5 to 15 cm., and there are no signs of right or left heart failure. He will be kept under medical surveillance, particularly watching for evidence of weight gain or other sign of impending heart failure.

SUMMARY

New advances in therapy permit us to do a great deal for patients with congestive heart failure. Rest, digitalis, diet, and diuretics are the main tools for achieving control. However, the degree of success in any given case is determined by the skill and manner in which these tools are used.

CARDIORESPIRATORY failure with kyphoscoliosis is of both anatomic and functional origin. The anatomic factors, small lung volume and thickening of the media of the vessels, are largely irreversible, while the functional resistances—hypoxemia and consequent high pulmonary blood flow, polycythemia, and hypervolemia—are amenable to treatment. Use of a mechanical respirator appears to be the best treatment for hypoxemia and hypercapnia, and the Drinker-type machine and the intermittent positive pressure apparatus are also effective. The respirators establish a breathing pattern with larger tidal volumes, which favor alveolar ventilation.

Hypercapnia precludes the use of enriched oxygen mixtures for hypoxemia. Drugs that cause depression of the central nervous system are also avoided. Congestive failure is treated with digitalis and diuretics, and salt is restricted, while venesection combats polycythemia and hypervolemia. When an acute infection occurs, antibiotics are used, and bronchodilators are employed to aid patients with asthmatic bronchitis. After recovery, gas concentration in arterial blood, erythrocytes, and venous blood pressure should be measured frequently to detect possible recurrence.

E. H. BERGOFSKY, G. M. TURINO, and A. P. FISHMAN: *Cardiorespiratory failure in kyphoscoliosis. Medicine* 38:263-317, 1959.

Regressive Electroshock Therapy in Chronic Schizophrenia, a Controlled Study

Preliminary Report

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REGRESSIVE ELECTROSHOCK TREATMENT (REST) has been recommended for patients suffering from chronic mental disturbance, particularly for those with chronic psychoneurosis¹ and chronic schizophrenia.² The present paper contains a preliminary report of results obtained three months after termination of REST given to a group of patients with chronic schizophrenic disorders. It should be noted that (1) this is the first attempt to perform a controlled study of REST; (2) the patients involved have a longer average duration of illness than those in previous published studies; and (3) Mecholyl tests were administered. The prognostic relationship of these tests to REST will be discussed in a separate report.

REVIEW OF PREVIOUS RELATED STUDIES

Previous reports about the therapeutic value of REST for chronic schizophrenia are contradictory. They are discussed by Glueck and associates² in a recent publication. The authors state (1) that the treatment procedure was inadequate in the patients reported on by Kennedy and Anchel,³ by Rothschild and associates,⁴ and by Weil⁵ and (2) that the patients selected by Garrett and Mockbee⁶ had too poor a prognosis. In 1948, Kennedy and Anchel³ treated 25 chronic schizophrenics with regression to the level of a 4-year-old child; of their patients, with an average illness duration of 4.5 years, 13 showed good improvement, 11 moderate, and 1 no improvement. Weil,⁵ in 1950, used a more drastic form of REST on his 18 patients. He reported 2 to be considerably improved, but both relapsed after seven and eight months, respective-

ly. In 1951, Rothschild and associates⁴ treated 52 patients with the predetermined number of only 28 electroshock treatments. Of their patients, with an average illness duration in men of 8.8 years and in women of 5.7 years, 36 per cent achieved considerable improvement, 21 per cent slight improvement, and 43 per cent remained unchanged. One year later, Garrett and Mockbee⁶ selected 30 of the most regressed and therapy-resistant schizophrenics, with an average illness duration of three to six years; the majority of their patients showed symptomatic improvement but no social remission. The best results with REST given to psychotic patients are reported by Glueck and associates,² who treated 100 cases between 1948 and 1953. Besides 74 schizophrenics, however, their study comprises 20 patients suffering with some variety of schizophrenic illness and 6 nonschizophrenics, yielding an average illness duration for the female patients of 6.3 years and for the men of 4.6 years. Results included 48 per cent recovered or markedly improved, 24 per cent improved, and 28 per cent slightly improved or unimproved. Twenty-two patients relapsed three weeks to twenty-seven months after discharge from the hospital, and 5 received a second series of REST; but the over-all evaluation of late results demonstrated a further shift in the direction of improvement, with 71 per cent of the patients functioning satisfactorily on the outside.

EXPERIMENTAL DESIGN

1. *Selection of Patients.* For the present study, conducted from July 1956 to October 1957, a total of 104 chronic schizophrenic patients from a state hospital population served as subjects. Their ages ranged between 20 and 55 years, with an average of 45.9 years for the 18 female and of 43.9 years for the 86 male patients. The dura-

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tions of their illnesses ranged between the extremes of three and thirty-five years, averaging 17.6 years for the females and 17.1 for the males. An attempt to differentiate between the schizophrenic subgroups was abandoned; the psychotic manifestations of most of these patients had shown marked alterations in the past and appeared mixed at present, causing previous changes of diagnoses and only controversial classification at present. All patients had a conspicuous onset of their psychosis, with mental excitement, assaultive or destructive behavior, and delusory and hallucinatory experiences; all of them still suffered with psychotic manifestations of varying degrees and mixtures. Most of them were judged as behaviorally and emotionally deteriorated; about a third of them had occasional or frequent urinary incontinence. All of them were ambulatory, and the greater half participated in undemanding work assignments and social activities within the institutional framework. The majority of them had received one or several course of Metrazol, electroshock, and/or insulincoma treatment; only for 26 of the 104 patients was no previous somatic therapy recorded.

Not included in this study were (1) schizophrenic patients who had not exhibited acute psychotic manifestation in their past or present, the so-called simple type; (2) patients who were suspected to have an organic component contributing to their schizophrenic reaction; and (3) schizophrenics who had undergone psychosurgery.

2. *Nature of Treatment.* The technic of REST, described in detail by Glueck and associates,² consists of electrically induced grand mal convulsions given 3 times daily until pronounced mental regression and neurologic evidence of altered cerebral function are reached.

In this study, a minimum of 19 and a maximum of 66 electroshock treatments were necessary to produce the prescribed level of mental and neurologic impairment; in the majority of cases, 25 to 35 treatments were sufficient. All treatments were modified through the regular use of Anectine, and barbiturates were given intravenously prior to the initial treatments in a few cases complicated by marked anxiety or resistiveness.

No severe complications occurred during the treatment and recovery period of these 52 experimental patients, with the exception of one death, the details of which are given in the case report.

3. *Controls.* Permission to administer the experimental treatment was obtained for 52 patients. A total of 13 separate experiments, aver-

aging 4 patients per experiment, were conducted, each one requiring six weeks for the administration of REST and for the return of memory, orientation, and neurologic function. The groups followed each other in three-week intervals, with consequent overlap. There were eight experimental groups of 4 patients each, 3 of 3 patients each, 1 of 5, and 1 of 6.

The experiments were performed in a structured environment, the patients being separated during treatment and initial recovery period from the general hospital population. This simplified their management and avoided undue fear among the other patients. The same psychologic factors were applied to all patients insofar as this could be ensured by separation of the subjects from the usual hospital environment and from external contacts by isolation on a single ward. Special care was provided by a small number of regularly assigned nurses and psychiatric aides.

Each experimental patient served as his own control, being appraised by a variety of devices (discussed below under assessment technics), both preexperimentally and again three months after the cessation of treatment. In addition, as a control on the effect of stimuli in the environment other than REST, each of the 13 experiments included a control group (of the same size as the experimental group) drawn from other patients with chronic schizophrenia. There were 52 control patients in all, matched with the experimental subjects as to sex and partially matched as to age and duration of illness. Complete correspondence on the latter 2 variables was not, of course, attained. The mean age of the treated subjects was 42.4 years (with a range of 41 years), and the mean age of the controls was 46.1 years (with a range of 33 years). The duration of illness averaged 15.4 years (with a range of 28 years) for the experimental subjects and 18.9 years (with a range of 36 years) for the controls. These factors receive further comment below in the discussion.

All patients, experimental and control, were informed that they would receive the benefit of concentrated treatment in a specific hospital unit, and an attempt was made to approach treatment and control patients with similar attention and attitudes. Naturally, the experience of mental regression to almost complete amnesia could not be duplicated in the controls, and arrangement of factitious treatment was not feasible.

4. *Assessment Technics.* Psychiatric effects as of three months after treatment were gauged by means of (1) the major criterion of the patient attaining or failing to attain discharge from the hospital, (2) gross changes in behavior as mani-

fested in testability or lack of testability on the Minnesota Multiphasic Personality Inventory (MMPI) and on the Shipley Institute of Living Scale of Intelligence (Shipley-Hartford), and (3) subtle changes in behavior as detected by the Wittenborn Psychiatric Rating Scales (Wittenborn). The crude use of self-report instruments (MMPI and Shipley-Hartford) was necessitated by the percentage of testable patients, which invalidated a comparative psychometric study of psychopathology.

RESULTS

In order of the assessment criteria, the results were as follows:

1. Recovery or social remission to the extent of accomplishing the discharge of a patient was not achieved during the observation period.

2. Changes in status during the observation period from untestable to testable (or vice versa) on the MMPI and on the Shipley-Hartford were found not to differ significantly between experimental and control patients.

3. Two Wittenborn protocols, preexperimental and postexperimental, were available on each subject. Hence the variable analyzed was the difference, "pre minus post," in total score on the two protocols. (The separate scales of the Wittenborn as well as various derived subscales were examined both on a priori grounds and by item analysis. All efforts pointed to total score as the most relevant indicant.) Since low totals on the Wittenborn indicate less pathologic behavior, positive values of the variable are in the direction of psychiatric improvement (as reflected by the Wittenborn).

In 8 of the 13 experiments, the average positive change on the Wittenborn was higher for the experimental subjects than for the controls; in the remaining 5 experiments, the controls yielded a higher average. Though the separate experiments were thus not entirely uniform, this experiment-treatment interaction was not significant. However, the main effect of REST was significant at the 5 per cent level. For the experimental subjects, the over-all average change was a positive 11.0. For the controls, the mean change was a positive 5.8. Thus the average REST effect, as gauged by Wittenborn total score change, was a positive 5.2 points, with a standard error of 2.6 points.

Alternatively, a more descriptive presentation of the effect of REST is possible in terms of the scope of individual patient variation. Three categories of total score changes on the Wittenborn may be defined as follows: (1) above 40, much improvement; (2) 20 to 40, some improvement;

(3) -20 to 0 to 20, slight or no improvement. With these categories in mind, an examination of the Wittenborn protocols revealed the following results: From the treated group of 52 patients, 3 showed "much improvement," 10 "some improvement," and 39 "slight or no improvement," which would indicate that 25 per cent of these patients responded positively to REST. From the control group of 52 patients, none showed "much improvement," 6 "some improvement," and 46 "slight or no improvement," which amounts to 11.5 per cent of these patients responding favorably.

CASE REPORT

A physically healthy male 30 years of age developed a fever of 104° F. eight hours after termination of his REST, with 30 treatments. He was mute and unresponsive and showed slight general spasticity. Respirations were 30 per minute; pulse rate was accelerated up to 140 beats per minute, blood pressure was normal, and skin color was good. His condition was maintained over thirty-six hours with antibiotics, fluid regimen, and routine nursing care. A spinal fluid examination showed normal dynamics, no cells, and total protein 34 mg. per cent. On the third day a marked paradoxical respiration with generalized suppression of breath sounds and total absence of breath sounds on the right side were observed. Attention to his airway did not bring improvement. X-ray of the chest showed no pulmonary infiltration. Bronchoscopy was done and a large piece of meat was removed from his trachea. After this, his respirations slowed down and his heart rate decreased but his temperature remained high. On the morning of the fourth day it had risen to 106.8° F. rectally, where it stayed for about one hour. An ice-bath lowered his temperature, which fell to a low of 95° and rose again to 97° F. The patient's condition deteriorated steadily. He became very cyanotic and almost pulseless during the episode of high fever and continued this way after it. He died in the early afternoon of the same day.

Autopsy showed no gross pathology of the inner organs. The lungs and tracheobronchial tree were unremarkable. The lungs had normal weight and normal gross appearance. The heart, weighing 500 gm., showed moderate hypertrophy of the left ventricle and mitrovalve changes compatible with an old and asymptomatic rheumatic valvulitis. External examination and section of the brain revealed no abnormalities other than congestion of the superficial vessels. Microscopic examination of sections revealed congestion of the cerebral vessels and

petechial hemorrhages in the cerebral white matter and to some extent in the cortex. About some of the small vessels were small numbers of mononuclear leukocytes and some phagocytes containing a light brown pigment. There were prominent petechiae in the hypothalamus. These are generally considered to arise on an agonal basis; however, the prominence in these sections suggested the possibility that they may have existed prior to the agonal state. The diagnosis was cerebral petechiae and congestion, with the cause of death hyperpyrexia, due to acute brain syndrome, in some way related to REST.

DISCUSSION AND SUMMARY

Some impressions about the 13 experimental subjects who improved through REST will be formulated here. Apparently younger age and consequently shorter duration of illness are as important indications for REST as for other forms of somatic therapy. The 3 patients who showed much improvement were 19, 21, and 28 years of age and had been psychotic for four, five, and five years. The age factor also played a role in the patients showing some improvement, since 2 of them were between 20 and 30 years and 3 of them were in their thirtieth year (the ages of the other 3 improved patients were 41, 50, and 51 years). In attempting to find some prognostic clues in their psychopathology, it was noted that (1) 4 patients had severe paranoid delusions in a fairly well integrated personality pattern, (2) 4 showed a strong affective component combined with their schizophrenic psychosis, and (3) 3 were known to have severe psychotic episodes with intervals of partial remission.

Of the 6 control patients who showed some improvement, 2 also belonged to the younger age

group (28 and 33 years of age) and both had a schizophrenic illness with episodic character. No particular reason for the mild improvement in the other 4 control subjects was apparent. Their ages ranged between 45 and 55 years and their durations of mental illness between 21 and 39 years. All of them were chronically delusional, occasionally disturbed, and moderately regressed.

It would be premature to arrive at conclusions about the effectiveness of REST in the treatment of these chronic schizophrenic reactions because only the immediate results are reported in this study. Relapses as well as further improvements can be anticipated. A follow-up study is under way, and the observation by Glueck and associates² of an increasing number of late positive results will be tested on this case material. However, it may already be stated that one-quarter of severely ill and therapy-resistant schizophrenic patients responded favorably to REST within an observation period of three months.

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Unilateral Polycystic Kidney Disease

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WHEN ONE SPEAKS OF renal polycystic disease, the usual connotation is that of the bilateral condition. Rarely, however, is a true case of unilateral polycystic disease of the kidney encountered, and it is with this condition that this discussion will be concerned. We will briefly discuss some of the literature concerning polycystic renal disease, emphasizing unilateral polycystic disease, and present a case report of a recent encounter with this disease.

Any discussion of polycystic renal disease, bilateral or unilateral, necessitates a definition of terms. Considerable confusion continues to exist in the literature due to a general failure to adopt a uniform nomenclature regarding cystic renal disease. Such haphazard terms as "multicystic" and "polycystic" are used as though they were synonymous. Only recently has improvement in this regard become evident in the literature. Ravitch and Sanford¹ have clearly differentiated unilateral multicystic disease from polycystic disease. Polycystic renal disease is characterized by normal fundamental renal architecture which has been compressed by cyst formation. In multicystic disease, there is no semblance of renal architecture in the affected kidney, cortical substance, papillae, or pelvis. Occasionally, a fibrous cord-like ureter has been observed. Spence,² in his review of 15 cases of unilateral multicystic renal disease with the addition of 4 new cases, points out some common characteristics of multicystic disease which aid in the differentiation of these two entities. The multicystic kidney resembles a bunch of grapes. It is practically devoid of normal architecture. The ureter may be patent but is frequently atretic, and the blood supply may be variable. Grossly, the true infantile and adult polycystic kidneys offer a semblance of the kidney, and, microscopically, renal tissue is noted. The infantile polycystic kidney is sponge-like. The simple and multilocular cystic kidneys are normal except for the involved portions. The

following features are noted in unilateral multicystic disease: (1) Symptoms are not characteristic and may be absent; (2) Urinalysis may be normal; (3) Intravenous urography may show absence of excretion of opaque solution on the involved side with normal excretion and normal pattern on the contralateral side; and (4) Cystoscopy may reveal absence of the ureteral orifice or ureteral obstruction on the affected side.

Polycystic disease, however, exhibits a wide variety of signs and symptoms. In his excellent monograph, Dalgaard³ presented a follow-up of 284 cases and presented the following data. The age range was 25 to 85 years. Uremia was the cause of death in 59 per cent of patients, cerebral hemorrhage in 13 per cent, heart disease in 6 per cent, and miscellaneous causes in 22 per cent. The symptoms and signs were pain and vague abdominal complaints in 59 per cent, occurring largely in women; renal colic in 64 per cent, prevalent in men; hematuria in 45 per cent, occurring with equal frequency in both sexes; and cardiovascular signs in 46 per cent, also appearing an equal number of times in men and women. Terminal uremia was noted in 44 per cent, palpable kidney tumors in 64 per cent, proteinuria in 75 per cent, and pyuria in 46 per cent. All four conditions occurred more often in women. The average age of death was 51.5 years of age for both sexes.

Simon and Thompson⁴ presented a twenty-year study of 366 cases from the Mayo Clinic from 1925 through 1944. This series consisted of 203 men averaging 55.5 years of age and 163 women averaging 44.5 years of age. In this group, 133 were asymptomatic, 130 had dull pain, 33 had colic, 115 had gross episodic hematuria, 77 had infection, and 28 had gastrointestinal symptoms associated with terminal uremia. Fifty-five per cent were hypertensive, and 51 per cent had palpable abdominal masses.

An understanding of the comparative rarity of the unilateral polycystic condition is given by Burman and Oweida,⁵ who cite Bell's⁶ report in Herbut's⁷ text classifying renal cystic disease and reporting 44 cases in 22,393 autopsies, 40 of

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which were bilateral and 4 unilateral. In this series, there was only 1 patient between 4 months and 25 years of age and the disease was unilateral in that patient. Oppenheimer and Narins⁸ reported another case of unilateral disease in a man 52 years of age. Upon reviewing the literature, they found an incidence of 2 cases of unilateral disease in 88 cases of polycystic disease encountered at autopsy, 2 cases of unilateral disease and 14 of bilateral disease in 10,177 autopsies, 4 cases of unilateral disease and 11 of bilateral disease in 4,903 autopsies of infants, and 11 cases of unilateral disease and 64 cases of bilateral disease in 32,360 autopsies and a variation of unilateral to bilateral disease in from 4 to 18 per cent.

Dalgaard³ gives a frequency of 143 cases of bilateral disease in 98,000 autopsies, or 1 in 773. Weinberg and associates⁹ cite the work of Bugbee and Wollstein,¹⁰ who studied the urinary tract of 4,903 infants at necropsy and found a total of only 4 cases of unilateral cystic renal disease, an incidence of 0.1 per cent. Other case reports are those of Mathe,¹¹ Howze and Hill,¹² and Branham.¹³ The latter reported a case of a young woman with a solitary polycystic kidney who maintained a normal life for over eight years despite serious symptoms of cardiovascular-renal decompensation. She became pregnant, delivered, and lived four years postpartum. More reports are those of Carlson,¹⁴ Moore and Buchert,¹⁵ Rall and Odell,¹⁶ Menashe and Smith,¹⁷ Eto and Mackey,¹⁸ and Schwartz.¹⁹

Not only does confusion exist in the nomenclature, but scattered through the literature are assertions that unilateral polycystic kidney disease is not an actual entity and does not occur. Some^{11,20} feel that the disease is always bilateral even though it may be manifested in only one kidney, because early and latent cysts in the opposite kidney may be overlooked and because cyst formation may be much slower in one of the kidneys than in the other.

Nothing is known of the exact pathogenesis of polycystic kidney disease except that it is hereditary.³ It is claimed that the mode of inheritance is a recessive and nonsex-linked gene. The most widely accepted theory concerning the origin of the polycystic kidney is that of Hildebrandt.²¹ His theory, formulated in 1894, assumes incomplete fusion of the terminal elements of the ureteral bud and the tubules of the kidney. The nephrons cannot dispose of their fluid content and cystic atrophy occurs. Kampmeier²² has questioned this theory and attributed the formation of cysts to incomplete involution and subsequent cystic degeneration of earlier

mesonephric tubules. He demonstrated that these earlier generations of uriniferous tubules and their glomeruli normally become detached from collecting ducts and joined collecting ducts of a higher order. An early overproduction of uriniferous elements was then followed with a degeneration and reduction of the number of these elements. Some of these renal tubules remain temporarily as cysts, and it was suggested that the persistence of a large number of these cysts might cause polycystic disease. Huber²³ believed that the improper proliferation of the metanephrogenic anlage occurred, preventing normal union of its elements with those of the developing ureter and pelvis. In 1948, Goodyear and Beard,²⁴ speaking of the multicystic variety, suggested that the disorder results from congenital ureteral obstruction, the cysts representing dilated and anomalous major and minor calyces. It is felt that the multicystic situation is nonhereditary as opposed to the background of the polycystic condition. Further elaboration on the etiology of polycystic disease has been given by others more recently, notably, Alderman²⁵ and Frazier.²⁶ The latter feels that, while the relationship between multilocular cysts, polycystic disease, and large solitary cysts of the kidney is not clear, it would seem logical to assume that they may represent different manifestations of a common etiologic factor, which is probably congenital in nature.

An interesting report is that of Lambert²⁷ who reconstructed 2 infant and 5 adult polycystic kidneys and found that, in the infant, the majority of the cysts were distended nephrons or enlarged portions of nephrons that were separated from their excretory tubules, thus forming a closed system not communicating with the pelvis. In the adult, some of the cysts were similar to the infant type, but many were tubular in origin and were connected to the tubules in the renal pelvis and still retained some functional ability. For this reason, he expressed the opinion that patients with the adult type were able to live beyond infancy.

Perhaps of some interest is Dalgaard's³ statement concerning anomalies associated with polycystic renal disease. "... the association between polycystic kidneys and polycystic liver is a biologically real one, and, provided that the association between polycystic kidneys and aneurysms of the basal arteries of the brain were also biologically real, it would be natural to set up the hypothesis that a single gene is the cause of the syndrome of malformations, namely, polycystic kidney, polycystic liver, and congenital aneurysms of the basal arteries of the brain with

wide variation, particularly in the latter components." He states that there is no relationship between polycystic kidneys and the von Hippel-Lindau syndrome or hypernephroma.

TREATMENT

With the lack of unity that exists concerning nomenclature and etiology and whether the entity exists separately or not, one would anticipate disagreement in regard to the proper mode of therapy. Indeed, the literature disagrees in this respect but to a lesser degree. Dalgaard² points out that the Rovsing operation of multiple puncture of cysts is one method of choice, and he cites cases of patients who were not treated who had a mortality rate twice that of those treated with Rovsing's operation. Further, he feels that the results of Rovsing's operation and of nephrectomy are approximately the same with respect to mortality. In general, he feels that, in an effort to save renal tissue, conservative treatment is the treatment of choice and that surgery is indicated only if the pain becomes too severe. Ravitch and Sanford¹ cite the following indications for Rovsing's operation: (1) diminution of renal function, (2) persistent pain, (3) intracystic hemorrhage, (4) progressive elevation of blood pressure, (5) limited renal involvement, and (6) poor results of medical treatment. Other modifications of Rovsing's operation consist of decapping cysts and splitting the kidney down to the pelvis and suturing the resulting flaps to tissue just under the skin in addition to decapping and evacuating the cysts. They feel that, if the patients are carefully selected and if the operation is performed early enough in the course of the disease, this type of surgery unquestionably prolongs life. Nephrectomy should be done for coexisting malignancies, pyonephrosis, tuberculosis, irreversible hydronephrosis, intractable diffuse pyelonephritis, or extensive calculus disease provided the function of the opposite kidney is adequate as revealed by the concentration of nonprotein nitrogen. Destructive lesions limited to either pole of the polycystic kidney may be relieved by partial nephrectomy. Speaking of the disease in newborns and infants, Bugbee and Wollstein¹⁰ favor nephrectomy if the opposite kidney functions well, as do Good-year and Beard.²¹ In 1957, Brieker and Patton²⁸ presented a series of 4 cases of bilateral polycystic disease in which serial studies were done. One patient underwent bilateral surgical decompression, unilateral decompression was performed in another, the third was operated on but no decompression was done, and the fourth was treated conservatively for six months. It was

concluded that surgical decompression of the kidneys did not improve renal function satisfactorily, and the procedure was considered possibly detrimental.

CASE HISTORY

Mrs. A. O., a 54-year-old farm wife, was admitted to Trinity Hospital in Jamestown on August 7, 1958, with complaint of hematuria. Past history disclosed that she had had a hysterectomy, a thyroidectomy, and some previous rectal surgery and, in January 1958, a left heminephrectomy had been performed elsewhere because of a solitary cyst in the upper pole of the left kidney. She had done well since her kidney operation but had noted some blood in her urine for a short time. On the day of admission to the hospital, she noted frequency and pain over her lower abdomen and was only able to pass a small amount of urine with each voiding. She was weak, dizzy, and nauseated but did not vomit. Her blood pressure was 144/99, and her temperature was 98.4° F.

Physical examination disclosed moderate dehydration and slight cyanosis with some distention of the lower abdomen. The initial catheterized urine specimen showed that the urine was packed with blood. The initial hemoglobin was 11.2 gm. with an erythrocyte count of 4,200,000 cells per cubic millimeter. The following day her hemoglobin had dropped to 9.8 gm., and it was necessary to give her 1 unit of blood. Two days later, a biopsy and fulguration of two polypoid lesions of the right dome of the bladder were done. The postoperative diagnosis was polypoid tumor of the bladder with hemorrhage. The pathologic diagnosis was nonspecific inflammation. Following additional transfusions, the patient was discharged on August 16 with a final diagnosis of hemorrhage from an inflammatory lesion in the bladder.

She was readmitted to Trinity Hospital on August 24, 1958, again experiencing gross hematuria. Again, the physical examination was essentially unremarkable. The urine was grossly bloody, and the hemoglobin was 13.6 gm. Hematologic consultation was obtained and ruled out the possibility of undiscovered blood dyscrasia. Cystoscopy was performed on August 25, and bleeding was discovered in the site of the previous fulguration, and, in addition, some polyps of the bladder neck were fulgurated. On August 26, the patient was again transfused, since her hemoglobin had dropped to 10.5 gm. On September 3, she underwent cystoscopy for the second time. Recurrent papilloma of the dome of the bladder was diagnosed postoperatively, and a resection of this area was advised. On September 6, an intravenous urogram was done, which showed that the renal collecting system on the right was normal in appearance, but, after approximately twenty minutes, the lower calyceal group of the left side was visualized and showed considerable distortion and clubbing of the remaining calyces. At no time were the middle and superior groups visualized. On September 8, the patient was taken to surgery with a preoperative diagnosis of possible carcinoma of the bladder. The bladder was opened through a suprapubic midline incision, and a segmental resection of the dome of the bladder was performed. Again, the pathologic diagnosis was ulceration of the bladder mucosa with an extensive subacute chronic nonspecific granulomatous reaction.

Postoperatively, the patient did well until the afternoon of September 22, when she suddenly hemorrhaged from the bladder again and went into deep shock, her

hemoglobin dropping from 11.4 to 9.8 gm. She was given supportive therapy consisting essentially of vitamin K₁ oxide, rutin, vitamin C, ACTH, and multiple transfusions.

She responded well to this treatment, but, from September 27 to October 3, she bled on several occasions. Cystoscopy was done immediately following these episodes without success in determining the exact location of the bleeding point. Late in the evening of October 3, the patient bled again and was taken to surgery where it was discovered that she was bleeding from the left ureter. During the next few days, she bled briefly on several occasions. On October 6, she bled continuously all through the night, and it was felt that surgery should be undertaken immediately in order to remove the remaining portion of the left kidney, since this was evidently the site of the hemorrhage.

The following morning, a left nephrectomy was attempted but was unsuccessful, since any attempt to dissect about the upper pole produced profuse uncontrollable hemorrhage. The patient went into severe shock and became pulseless with no discernible blood pressure and, at times, no discernible heart beat. Due to her extremely poor condition, it was felt necessary to pack the wound as tightly as possible and close the wound over the pack. Again she responded to supportive therapy and transfusions and did fairly well until the evening of October 11, when she experienced a paralytic ileus and some pneumonitis of the base of the left lung. A Cantor tube was inserted, and, on the evening of October 12, the patient suddenly deflated, exhibiting a hard mass in the left flank which was felt to be a large hematoma. She again went into severe shock. She was supported through that night and was returned to surgery the following morning where, through a left rectus incision, the renal vessels were ligated and the remaining portion of the left kidney was removed with considerable difficulty. The patient tolerated the procedure rather well.

Grossly, the specimen was approximately 12 x 7 x 6 cm. with a large central cavity measuring about 7.5 cm. in length and 5 cm. in width, which contained yellowish-brown necrotic tissue. On cut section, the kidney contained many small cysts near the cortical medullary junction, which measured approximately 1 cm. in diameter. All of these cysts contained coagulated necrotic tissue. The calyces were dilated, and the pelvis was entirely replaced by a hemorrhagic cyst. Microscopically, there were multiple abscess formations in the renal cortex. The renal stroma was extensively infiltrated with polymorphonuclear leukocytes. The glomeruli were relatively undeveloped. There were numerous scarred and hyalinized glomeruli and marked hyalinization of Bowman's capsules. The grossly dilated kidney pelvis revealed hyalinized connective tissue in the wall of the enlarged cystic spaces and in the smaller cysts which was surrounded by an embryonic type of connective tissue which was also noted in the cortex of one of the sections. The final pathologic diagnosis was congenital cystic kidney with enlarged peripelvic cysts complicated by a subacute pyelonephritis and hemorrhage.

She progressed satisfactorily and was discharged on October 25 in good condition and has remained so to date.

COMMENT

Although some have questioned the possibility of unilateral polycystic kidney disease existing as a separate entity, the literature contains many

apparently verified reports of this condition, and we feel that the entity does exist. In our perusal of the literature, we found that much of the confusion is due to overlapping and misuse of the terminology used to describe the various pathologic pictures seen, especially with regard to the terms "multicystic kidney" and "polycystic kidney." Pathologically, these two entities are quite different. The treatment advised for polycystic renal disease consists essentially of three procedures: namely, the Rovsing operation of cyst decompression, heminephrectomy, and nephrectomy. The case report included in this paper describes our only experience with unilateral polycystic kidney disease and is apparently a case in which heminephrectomy was done for a simple cyst of the upper pole. Subsequent formation of additional cysts resulted in the need for a total nephrectomy.

SUMMARY

1. The literature concerning polycystic kidney, unilateral polycystic kidney, and unilateral multicystic kidney has been reviewed.

2. The confusion existing in the literature with regard to the proper use of the appropriate nomenclature has been commented upon.

3. The evident differences between polycystic renal disease and multicystic renal disease have been reviewed.

4. Some of the modalities of treatment and the results of such treatment have been discussed.

5. The case report of our experience with unilateral polycystic kidney disease has been presented.

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POSTOPERATIVE water and electrolyte balances in newborn infants are significantly different from those of adults.

Balance data for sodium, potassium, chloride, nitrogen, and water were obtained postoperatively for 15 infants undergoing surgery within the first four days of life and for 5 control infants of comparable ages. The urine volumes of operated infants were equal to or greater than those of the controls, suggesting that the newborn has a diminished capacity to conserve water postoperatively but a normal capacity to dilute urine. No water retention was observed.

Although concentrations of sodium and chloride on the serum remained normal or increased slightly after surgery, urinary excretion of sodium and chloride was equal to or greater than that of the controls. Infants with losses from gastrointestinal suction had no capacity to reduce renal excretion of these ions while sustaining large extrarenal losses.

Postoperative losses of potassium and nitrogen in urine were proportional to those usually observed in adults.

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Desensitizing Factors in Cases of Migraine

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A GREAT STEP FORWARD in medicine would be taken if we physicians could learn why persons who suffer from episodic disease, such as migraine, epilepsy, asthma, fibrositis, arthritis, peptic ulcer, or psoriasis every so often will get well and then be free of attacks for months or years. Then the disease will return, and, even when the sufferer is an observant physician, he will be unable to guess why the distress left him or why it came back. For sixty years, I have been subject to migraine and fibrositis, but why the scotomas or the attacks of muscle pain come or stay away, I only occasionally can say. That the mechanism that produces the migrainous spells must, at times, be desensitized or thrown out of action is indicated by the fact that, in my free intervals, the most trying and fatiguing stimuli, which ordinarily should produce a scotoma, have no effect. For two months or more, the usual triggers will not work. As a highly migrainous army officer said to me, "During the most trying experience of my life, when, in England, I was responsible for sending a constant stream of supplies to the armies invading the French beaches, and, for six weeks, I hardly had time to take off my clothes, why was I free from my terrible headaches?" Hundreds of patients have described this same puzzling phenomenon of freedom from migraine during serious crises in life when there was much strain, overwork, fatigue, anxiety, annoyance, or sorrow. Surely, we physicians should be studying this phenomenon of desensitization because, if we should ever understand its mechanism and learn to use it—perhaps by inducing a mild hepatitis or changing some function of the glands of internal secretion—we could give millions of people grateful relief from much suffering.

Hormonal factors. That one of the desensitizing

EDITORIAL NOTE

Migraine is a condition that makes life almost unbearable for many people, and when an author with the experience of Dr. Alvarez records in detail his experience with this condition, it is felt that this experience should be shared with others who are dealing with the problem of migraine. Certainly this paper will be of interest to anyone interested in the problem of pain.

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ing factors is hormonal is indicated by such facts as that some migrainous girls lose their spells when they start menstruating, and many women remain well during a pregnancy or part of a pregnancy. One of my women patients, who for most of her life has had a severe and intractable type of migraine, was free from headache during the last five months of a pregnancy. The fact that she got a terrible sick headache *two days* after she delivered suggested that the desensitizing mechanism was a nervous rather than a hormonal one. In support of this idea was the fact that the only other time she "went free" was during World War II, when, for six months, she enjoyed teaching for half days in a university. Later, when she tried teaching full time, her headaches returned.

That the explanation for the relief of headaches with pregnancy is not simple is shown by the fact that some women lose their migraines while carrying one child but not while carrying another. The sex of the child is not the determining factor. Curiously, also, some women go free during only a few months of a pregnancy, while others have the worst type of migraine during their pregnancies. One girl at the age of

15, a year before she started menstruating, began to have a mild spell of migraine every three or four months. This sequence continued until she was 22 when, with her marriage to a doctor, the migraines disappeared. After that, migraine came only during the first five months of each of her four pregnancies—with one exception. She had one very severe spell a few weeks after her second confinement while she was nursing her infant. This spell came without any obvious cause.

As is well known, many a woman will be free from attacks of arthritis or peptic ulcer during a pregnancy.

Migraine commonly clears up when the patient is aged 20 or 30. Girls who suffer from migraine will sometimes go free when they reach the age of 20. One such girl, after the age of 20, went free for a few years. Then her sick headaches returned without obvious reason. Another girl, who began with her vomiting spells at 8, soon got over them and went free until the age of 37, when the storms returned. Then they were always menstrual. It is hard to explain a case like this on a physiologic basis. Why, also, should many men have severe migraine in their youth and then go free in their late twenties or early thirties? And why should an occasional man keep getting his severe headaches into his sixties? Why should an occasional man go free for thirty days at a time and then, each month, get a sick headache?

Periods of relief lasting a year or more. One of my women patients used to go into spells in which every night for a month she would get a severe sick headache. Then she would be free for a year. This cycle was repeated several times without obvious cause.

The good effects of hard work. Curiously, hard work with an escape from boredom will sometimes give a migrainous person a period of perfect relief. To illustrate: a physician, early in World War II, gave up a big practice to join the Navy and was sent to a graduate school for a refresher course. There, with only a half-dozen lectures to attend each day, he began to suffer from frequent and severe sick headaches. He and I struggled to find some explanation for this flare-up in his previously mild type of migraine, but we failed. Later, he wrote me from a Navy hospital to say that he was "cured" the day a ship arrived full of wounded men and he had to go hard to work. Evidently, at the medical school, he had not had enough to do and had felt a letdown after his busy practice.

Effects of accidents. Remarkable are the many cases in which a serious accident ushers in a long period of relief from migraine. Thus, a woman lost her migraines after an auto accident in which her legs were broken. A hard working doctor's nurse of 55 who, for years, had suffered from frequent attacks of severe migraine, went free for a year after she fell and broke her hip. That the relief was not all due to stoppage of work was shown by the fact that, as soon as she was out of the hospital, she was back on crutches running the office. A strange story is that of a patient of mine who was apparently cured of his migraines by a fracture of his skull! The Lippman's¹ described a similar case. In another case, a long period of relief followed the breaking of a leg.

Hemorrhage of fever. Several of my patients with a duodenal ulcer lost their migraine for six months or more after a hemorrhage. In the old days, a spell of typhoid fever used to "cure" migraine.

Disease of the liver and jaundice. Many physicians and laymen think that disease of the liver or gallbladder causes migraine, but I have much evidence to combat this view. On studying 215 cases of liver disease, Dr. Carl Morlock and I² confirmed the fact, long known to physicians, that the coming of jaundice will usually give the person relief from migraines for some months. In our series of 431 cases, there was twice the incidence of migraine among the controls, with an apparently normal liver, as among the persons with definite cirrhosis or hepatitis. Dr. Arthur Hertzler once told me that the only time in his life when he was free of migraine was during the year following an attack of jaundice.

A most instructive case was that of a patient of mine who was completely relieved from his ordinarily frequent migraines while he was coming down with a hepatitis and *before the jaundice arrived*. This showed that his respite was not due to the presence of an excess of bile in his blood but to the disease in the liver. This was shown me again by a migrainous woman who became severely jaundiced while taking chlorpromazine. With much bile in her blood, she went on with her headaches as usual. But, curiously, another woman who became jaundiced from taking Atabrine told me she remained free of migraine for a year!

In my long experience, I have never seen the removal of a diseased gallbladder permanently cure migraine, but, in some cases, it brought relief for many months, and, in one case, it gave

relief for six years. Then the migraine with abdominal pain returned just as before! A young woman lost her migraines for a few years after the removal of her appendix because of a syndrome indicating a "subacute appendicitis." Then the headaches returned unchanged.

Electroshock treatments and operations. I have seen a few overly tense and mildly psychotic patients lose their migraines for many months after they had had a few electroshock treatments for a depression. Others were helped but for only a short time. Curiously, one woman was relieved of severe migrainous headaches for a year after a bilateral splanchnicotomy, which was performed with the vain hope of relieving a constant abdominal pain for which no local cause could be found. I know of 5 cases in which a lumbar sympathectomy, performed to relieve hypertension, put a stop to the patients' sick headaches for months or a year. In some of these cases, the blood pressure did not stay down, which showed that a *reduction of pressure* was not the cause of the improvement noted.

The effects of fright. Edward Living³ quoted Labarraque, who told of a woman who, for years, had suffered from a severe and frequently recurring migraine. One day as she felt an attack coming on, she accidentally set fire to her cap and burned her forehead. The fright she suffered not only blocked the oncoming spell but ushered in a period of relief that lasted for some years!

Migraine absent during certain times of year. I have seen a few cases in which a person was always free of his or her severe migraines during the summer months.

Freedom during middle age. A large percentage of migrainous persons are free or practically free from headache in their middle years from 30 to 50 or so. After 50, the headaches may return for a number of reasons, such as much strain in life, hypertension, or cerebral arteriosclerosis.⁴

Influences of little strokes. In the paper just referred to,⁴ I tell of several cases in which a little stroke appeared to cure the person's migraine. For instance, a patient of mine, a man, had terrible sick headaches until the age of 61, when a stroke wiped out permanently his tendency to migraine.

Migraine often fails to stop at the menopause. Most of us think of migraine as stopping at the menopause, but, in many cases, it doesn't, and, in some cases, it becomes worse at that time or even begins at that time.

Operatively-induced menopause. Some sur-

geons think that a complete hysterectomy will cure migraine, but it rarely does. In every one of my last 113 cases in which a home surgeon had induced a menopause, the woman said she was no better. Many said they were worse after the operation.

Blindness appears usually to bring freedom from migraine. My blind patient, Mrs. Edna S. Sollars, once corresponded with most of the superintendents of schools and centers and workshops for the blind in the United States and found that, in their experience, migraine is a rare complaint among the blind. Among 633 blind adults, only 2 had reported having sick headaches. In one of these cases, the headaches stopped the day the woman lost her sight. Only 8 blind children among the 2,623 reported on appeared to be having migraine, with spells severe enough so that the school nurse knew about them.

One wonders if a lack of stimulation of the visual center by light can lead usually to a cessation of the migraines. It is not a sure cure because I know a woman who has been having severe sick headaches for fifty years, since her eyes were enucleated at the age of 20, and another woman who has had sick headaches all of her life in spite of the fact that she was born blind.

Insanity may put a stop to migraine. In 1886, G. H. Savage wrote that when a person with severe migraine goes insane, he or she loses the headaches. When Mrs. Edna Sollars wrote to the heads of a dozen mental hospitals in regard to this, they all said that, so far as they knew, their patients rarely complained of migraine. Mildly psychotic persons with their stormy emotions often suffer severely from migraine.

SUMMARY

A great step forward would be taken if we could learn why persons suffering from episodic diseases, such as migraine, asthma, fibrositis, peptic ulcer, or psoriasis, commonly have long periods of freedom from their trouble. During these periods, the mechanism that causes attacks appears to be thrown out of action, so that then even great strain or fatigue or distress will not start a spell.

There appear at times to be hormonal factors at work. Pregnancy can relieve migraine or arthritis, or it can heal a peptic ulcer. But, in many cases, pregnancy does not help. In one pregnancy, migraine may be relieved while, in the next, the spells will go on either unchanged or worsened.

Migraine commonly clears up by the age of 20 or 30. It can return at the age of 50.

Migraine can be made worse by boredom or a letup of strain.

Migrainous persons can be "cured" for months or a year by an accident, a gastric hemorrhage, an attack of fever, or a hepatitis. Relief appears to be due to the hepatitis rather than to the excess of bile in the blood. Migraine can be "cured" by electroshock treatments, by a lumbar sympathectomy, by a fright, by a stroke, by blindness or by insanity.

In 113 consecutive cases, migraine was not stopped by a hysterectomy, and often it is not stopped by a natural menopause.

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Book Reviews on Pain

JOHN S. LUNDY, M.D.

CHICAGO

Lectures on the Interpretation of Pain in Orthopedic Practice

ARTHUR STEINLEER, M.D., 1959. *Springfield, Ill.: Charles C Thomas*. 733 pages. \$18.50

This book represents the experience of an authority of many years' experience on the subject at hand. As the author says, "The principal objective of these lectures on pain in orthopedics rests in the hope that they may be a help in taking advantage of the state of reversibility of pathological conditions by recognizing them early enough." The subject is completely covered in 16 chapters in the form of lectures.

The book is well illustrated and is printed on excellent paper. The text is easily read. The subject index is extensive and another index is devoted to authors' names. A bibliography is appended to each section of the book. More books of this type are needed for the general dissemination of knowledge about pain, for it is only by thorough investigation of the problem of pain as it arises in each specialty that a broadening of information on this problem will come about.

Pain and Itch: Nervous Mechanisms

C. F. W. MOULSTENHOLME and MAEVE O'CONNOR, *Editors*, 1959. *Ciba Foundation Study Group No. 1*. Boston: Little, Brown & Co. 120 pages. Illustrated. \$2.50

Twenty scientists, including 1 from the United States, took part in a meeting on March 10, 1959, in honor of Professor Y. Zotterman of the Royal Veterinary College in Stockholm. The subject of the meeting was the nature of pain. This volume includes the papers and discussions of that meeting. Fortunately, the participants also included the subjects of itch and tickle, which can be regarded as being reasonably close to pain itself.

This small book greatly impresses the reader with the

great amount of research that has been done on the various nervous mechanisms in a great variety of animals. For those who are particularly interested in the explanation of some of the present phenomena of sensation, the book should be a great pleasure. It is indexed and contains an extensive list of references. The volume is recommended to those who are curious about the sensations they experience and how to explain them—even to such an ephemeral sensation as that experienced when a fly walks over one's skin.

Clinical Applications of Diagnostic and Therapeutic Nerve Blocks

JOHN J. BONICA, M.D., 1959. *Springfield, Ill.: Charles C Thomas*. 354 pages. \$8.75

Dr. Bonica says, "I have attempted to present the subject (in a concise monograph) in the hope that I may encourage even the busiest of practitioners to become acquainted with it and consequently to use it to the advantage of the patient. The aim of the book, then, is to present the evaluation of various nerve block procedures as diagnostic and therapeutic tools. A serious attempt has been made to present an objective assessment of the indications, effectiveness, advantages, as well as the disadvantages, complications, and limitations of these techniques."

Prominent in the book are the discussion and illustration of the nervous system, including the spinal and cranial nerves and the sympathetic and other autonomic nerves. Attention is given to the various drugs in use, and the description of techniques is supplemented with pictures, roentgenograms being used to show placement of needles.

The table of contents is detailed, and the index is extremely thorough. About 150 selected references are listed. The book is written in a clear style and printed on good paper. It should serve its stated purpose well.

Treatment of Gastritis, Ulcerlike Pain, and Ulcer Syndrome

Clinical and Roentgenographic Evaluation of New Medication

HENRY M. FEINBLATT, M.D., F.A.C.P.

Brooklyn, New York

PEPTIC ULCER is the most common organic disease of the gastrointestinal tract. An ulcer either has developed or may be expected to develop in approximately 5 to 10 per cent of the population. The medical treatment of this condition has general acceptance, and various drugs have been found effective in ulcer therapy.¹

The purpose of this report is to submit a clinical and roentgenographic evaluation of a new combination of standard ingredients, one of which—bismuth subnitrate—appears to give superior results because of its unique processing by fine trituration and minute particulation.

Numerous articles have appeared in European literature on this new formula, which is widely used there in the medical treatment of peptic ulcers and has recently become available to physicians in the United States.

The composition of the Romach (Roter) tablet is as follows:

Bismuth subnitrate (specially processed for fine trituration and minute particulation)	350 mg.
Magnesium carbonate	400 mg.
Sodium bicarbonate	200 mg.
Frangula	25 mg.
Calamus	25 mg.

LITERATURE

In a recent statistic analysis of 155 cases of peptic ulcer compiled from the European literature, Kupersmith² reported immediate relief of pain in 92 per cent of the cases treated with the Romach formula. After treatment was continued for three to six weeks, there was an average weight gain of 7.9 lb., and occult blood disap-

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EDITORIAL NOTE

The author of the paper, "Treatment of Gastritis, Ulcerlike Pain, and Ulcer Syndrome: Clinical and Roentgenographic Evaluation of New Medication," reviews the European literature on this question. In a study of 155 cases of specific ulcer, favorable results were obtained from the use of Romach tablets (Roter tablets in Europe), and relief was noted within three to six weeks in a high percentage of cases. The author has a series of 35 ambulatory patients who have been so treated. The results have been desirable with no untoward effects. This favorable outcome he attributes to the specially processed, finely trituated, and minutely particulated bismuth subnitrate contained in the tablet in question in combination with standard antacid agents.

Gastritis, ulcerlike pain, and the ulcer syndrome are seen so frequently that any satisfactory method of treating the triad should be welcome. For this reason, we are presenting Dr. Feinblatt's paper on the subject in the hope that it will be helpful, which we think it will.

JOHN S. LUNDY, M.D.

peared from the stools in all of 6 cases in which it was found. Eventual roentgenologic healing of the ulcer was reported in 81 per cent of cases.

Important features of the treatment, as reported in Europe, included its effectiveness for ambulatory patients, the long duration of the pain relief, correction of gastric hyperacidity, and absence of acid rebound side reactions.

The preceding results were considered sufficiently encouraging to justify an impartial clinical and roentgenographic evaluation.

RATIONALE

The antacid combination of magnesium carbonate and sodium carbonate is so well known that it requires no further comment. The former has a slow but prolonged action with considerable combining power for acid;³ the latter, a prompt action of brief duration.⁴ Together they provide both immediate and lasting antacid action with corresponding relief of pain and gastric hyperacidity.

The purpose of the small amounts of frangula and calamus is to offset the constipating effects of bismuth. The medication is not laxative.

Although salts of bismuth have been used extensively for their protective action⁵ on the ulcerated stomach or duodenal mucosa, the fine trituration and minute participation of the bismuth subnitrate (Roter process) in Romach tablets represents a definite innovation.

In artificially induced ulcers in guinea pigs and rats, histologic sections have shown that Roter processed bismuth subnitrate adheres closely to the damaged tissues and also encourages epithelization of the ulcer.⁶ Gastric ulcer walls were found to retain 4 times as much bismuth as compared with use of the *National Formulary's* ordinary bismuth subnitrate, and there was greater reduction of acidity.⁷

SCOPE OF INVESTIGATION

The purpose of this study was to determine the efficacy of Romach tablets in cases of gastritis, ulcer syndrome, and ulcerlike pain and the presence or absence of side effects or toxic manifestations resulting from the medication. To this end, clinical observations and roentgenographic examinations were performed.

The group studied was comprised of 35 ambulatory patients, 21 males and 14 females. Ages ranged from 16 to 78, with an average age of 48. Weight ranged from 119 to 252 lb., averaging 162 lb. Height ranged from 5 ft., 2 in. to 6 ft., 1 in. and averaged 5 ft., 7 in.

The diagnoses included duodenal ulcer in 9 patients, gastric ulcer in 2, duodenal spasm in 4, and gastritis with ulcerlike pain in 20. The duration of the illness ranged from two months to fourteen years, with an average of three years.

The most satisfactory dosage schedule was 1 tablet four times per day taken after meals and the after-dinner snack, which is often required in cases of gastritis and peptic ulcer. The average patient felt better and received greater benefit with this regimen than with administration of

2 tablets three times daily. The duration of treatment ranged from one to six months, with an average of four and three-tenths months. Most patients found that relief was fairly prompt and continued the medication willingly.

A thorough physical examination was made in each case. A careful history was elicited with particular attention paid to gastrointestinal complaints. A fluoroscopic examination was given or a roentgenogram was made in each case for diagnostic purposes. Following this diagnostic survey, medication was prescribed in accordance with the standard schedule.

During the medication period, frequent clinical, fluoroscopic, and symptomatic reexaminations were made and recorded.

In another group, roentgenographic examination was made in order to compare the dimensions of the barium meal, to observe the stations to which the barium meal progressed at the end of ten minutes, and to make similar observations at the end of four hours. Comparisons made before with results during treatment were designed to bring out differences in motility, spasm, and diagnostic changes brought about as a result of the medication. Clinical results were also noted in this roentgenographic group.

CLINICAL RESULTS

The symptoms studied included epigastric pain, epigastric discomfort, heartburn, flatulence, gas bloating, anorexia, diarrhea, and constipation. The effect of the medication on each of these symptoms will be considered separately.

Pain. The most characteristic clinical finding in all types of stomach disorders is epigastric pain. It may last for a short time, or it may be persistent and gnawing. Since it is inconstant in each case and does not follow the same pattern from day to day, the time element is not considered a proper measure of relief. However, the severity of the pain is usually quite consistent and, therefore, was rated as absent, dull, or acute.

Pain was present in 22 cases. It was acute in 8 and dull in 14. Pain was relieved by the medication in 21 cases, or 95 per cent; completely in 18, or 82 per cent; and partially in 3, or 14 per cent.

Discomfort. Epigastric discomfort has many causes. It may be due to subliminal pain, unrelieved gas, gastric spasm, or any combination of these factors.

Discomfort was present in 27 cases. It was relieved by the medication in 23 cases, or 85

TABLE 1
SUMMARY OF SYMPTOMATIC RELIEF UNDER
MEDICATION WITH ROMACH

Symptom	Cases	Relieved	Complete relief	Partial relief
Epigastric pain	22	21 (95%)	18 (82%)	3 (14%)
Epigastric discomfort	27	23 (85%)	14 (52%)	9 (33%)
Heartburn	23	21 (91%)	11 (48%)	10 (43%)
Flatulence	22	17 (77%)	11 (50%)	6 (27%)
Gas bloating	22	16 (73%)	12 (55%)	4 (18%)
Anorexia	17	13 (76%)	—	—
Diarrhea	8	3 (38%)	0	3 (38%)
Constipation	10	2 (20%)	0	2 (20%)

TABLE 2
PROGRESS OF BARIUM MEAL IN TEN MINUTES

Station	Number of cases before treatment	Number of cases during treatment
Stomach	15	15
Duodenum	15	15
1st half ileum	14	11
2nd half ileum	1	2

TABLE 3
PROGRESS OF BARIUM MEAL IN FOUR HOURS

Station	Number of cases before treatment	Number of cases during treatment
Duodenum	1	0
1st half ileum	2	5
2nd half ileum	14	14
Ascending colon	14	13
Transverse colon	8	8
Descending colon	3	3
Sigmoid	1	1
Rectum	1	1

per cent; completely in 14, or 52 per cent; and partially in 9, or 33 per cent.

Heartburn. Sour regurgitation is often described as heartburn. It consists of an eructation of gastric juice into the throat and mouth, causing an extremely sour and acid taste.

Heartburn was present in 23 cases. It was relieved by the medication in 21 cases, or 91 per cent; completely in 11, or 48 per cent; and partially in 10, or 43 per cent.

Flatulence. This symptom was present in 22 cases. It was relieved by the medication in 17 cases, or 77 per cent; completely in 11, or 50 per cent; and partially in 6, or 27 per cent.

Gas bloating. This symptom was present in 22 cases. It was relieved in 16 cases, or 73 per cent; completely in 12, or 55 per cent; and partially in 4, or 18 per cent.

Anorexia. Poor appetite was a complaint in 17 cases. Under medication, improvement occurred in 13 cases, or 76 per cent.

Diarrhea. This symptom was present in 8 cases. The medication afforded partial relief in 3 cases, or 38 per cent.

Constipation. This symptom was present in 10 cases. Partial relief occurred in 2 cases, or 20 per cent. The medication is not laxative, and the result may be considered fortuitous.

The clinical results are summarized in table 1.

No toxic or side effects were observed in any patient. Routine chemical and microscopic examination of the urine was negative in all cases.

ROENTGENOGRAPHIC RESULTS

Gastric roentgenograms after the barium meal were made before and during the treatment in 15 patients. No significant differences in the individual or average measurements of gastric contents were observed.

Before treatment, the average measurement of stomach contents was 12.4 cm. horizontal by 11.7 cm. vertical; during treatment, 13.3 cm. horizontal by 11.0 cm. vertical.

Romach did not change the rate of gastric evacuation. Before treatment, the gastric contents measured 0.5 cm. horizontal by 0.3 cm. vertical four hours following the barium meal; during treatment, 0.7 cm. horizontal by 0.3 cm. vertical.

Roentgenographic studies of gastric contents during medication with Romach revealed no gastric effect. The stomach contents were not impelled more quickly forward by the medication.

Comparisons of gastrointestinal motility are made by measuring the progress of the barium meal in terms of anatomic stations. Table 2 shows the results after ten minutes and table 3 after four hours.

Roentgenographic studies of intestinal movements revealed no substantial laxative or retarding effect in the portion of the bowel filled during the first four hours after medication. There was some retardation of the progress of the barium meal in 5 cases, slight additional propulsion in 1 case, and 9 cases remained constant.

SUMMARY

A statistic study of 155 cases of peptic ulcer compiled from the European literature indicated favorable results with use of the Romach (Roter) formula for three to six weeks. Pain was relieved in 92 per cent of cases; there was an average

weight gain of 7.9 lb.; and occult blood disappeared from the stools in all of 6 cases in which it was found. Eventual roentgenologic healing of the ulcer was reported in 81 per cent of cases.

Our group consisted of 35 ambulatory patients with gastritis, ulcerlike pain, or ulcer syndrome. The medication provided prompt and effective relief of the symptoms of epigastric pain and discomfort, heartburn, flatulence, gas bloating, and anorexia in the great majority of cases. There were no toxic or untoward effects in any case.

The roentgenographic studies revealed no gastric effect and no significant laxative or retarding action in the intestines after the barium meal. Thus, symptomatic relief is afforded without altering the rate or scope of gastrointestinal motility.

Therapeutic results have been attributed most significantly to the specially processed finely triturated and minutely particulated bismuth

subnitrate contained in the tablet in combination with standard antacids.

The product is known in Europe as Roter tablets and are available in the United States as Romach tablets, supplied by Ror Chemical Company of New York City.

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(1911)



(1959)

In Honor of

Elexious Thompson Bell, M.D.

LEMEN J. WELLS, M.D.

Minneapolis

IN CELEBRATION OF Elexious Thompson Bell's 79th birthday, it is a privilege for his colleagues and friends to express their appreciation of him. Dr. Bell, emeritus professor of pathology, University of Minnesota, is a man of alertness, high standards, and special talents. His professional competence asserts itself in morphology, pathology, and experimentation. His productivity goes on and on. What a man!

He was born in Hatch, Missouri, on August 30, 1880. His father, John Henry Bell, was a country doctor in the saddlebag days, who rode horseback almost up to the time of his death at the age of 80. His mother, Appeline Coontz Bell, was a relative of Admiral Coontz. Of the 7 children in his parental home, all but 1 was graduated from the University of Missouri. His uncle, Lee Bell, was a physician.

Young "Tommy" received instruction at home from a maiden aunt and entered school at the age of 10. Later, at the high school in Monroe City, Missouri, he was at the top of his class.

During his student days at the University of Missouri, he gave serious thought to becoming a mathematician but gave up the idea after his mathematics professor told him that the salary would be small. He was elected to Phi Beta Kappa. He was granted

the B.Sc. degree in 1901 and the M.D. degree in 1903. He was a member of a medical fraternity, Phi Beta Pi.

His first specialty was in the field of anatomy. At the University of Missouri, he served successively as assistant instructor in 1902 and 1903, instructor from 1903 to 1907, and assistant professor of anatomy from 1907 to 1910. He studied at the University of Bonn in Germany in 1905 and 1906. In Bonn, he lived at a pension and improved his conversational German by making short speeches for his young German colleagues. At the University of Minnesota, he served for one year as assistant professor of anatomy and worked with Professor Thomas Lee in 1910 and 1911. He helped in attracting to Minnesota 2 anatomists, Dr. Clarence M. Jackson and Dr. Richard E. Scammon. He and Dr. Scammon became roommates in Minneapolis. He rented a house for the Jackson family in advance of their arrival, thus demonstrating the extent to which that family trusted his judgment.

At the University of Minnesota, he was made assistant professor of pathology in 1911. In addition to teaching and research, he carried a heavy load of performing autopsies and testifying in court as an expert medical witness.

In 1914, he was married to Cecile Porcher. One of their special pleasures was travel. Their 2 chil-

LEMEN J. WELLS is professor of anatomy, University of Minnesota.

dren, Elizabeth (Mrs. Richard Ogle) and Robert Thompson Bell, were graduated from the University of Minnesota. Robert is a mining engineer.

Dr. Bell became a tower of strength in the Medical School of the University of Minnesota. He was associate professor of pathology from 1916 to 1920 and then professor and head of the department from 1920 to 1949. In the 1920's, he, in collaboration with the late Professors Clarence M. Jackson and Richard E. Scammon, was an outstanding leader in developing a strong program of medical research.

His success is attributable in part to "horse sense" in research, to a friendly manner, to a fine sense of humor, to his intolerance of sham, and to his ability to counsel wisely. He has talent for adding a touch of drama to prosaic topics. He is skillful in expressions which bring listeners promptly to the heart of a problem—"Never buy a horse from a farmer who has only one horse for sale!"

He is perhaps most widely known for his *Text-book of Pathology*, 8 editions of which were published; for his monograph, *Renal Diseases*, which appeared in 2 editions; and for his studies of hypertension and of diabetes mellitus. His monograph, *Renal Diseases*, has been translated into Italian and into Spanish.

As emeritus professor of pathology, he continues his research at the University of Minnesota and also serves as consultant in pathology at the Veterans' Hospital in Minneapolis. He is preparing a monograph on *Diabetes Mellitus*.

He has given up his hobby of riding horses but has retained 2 hobbies—double acrostics and detective stories. In order to hold his interest, a detective story must be full of "blood and thunder."

Shortly before his retirement in 1949, his friends and colleagues expressed their admiration and best wishes. Excerpts from three of the many letters may be cited.

Ambrose J. Hertzog, New Orleans: . . . When we brought to you our little Arbeits for possible publication, you not only made presentable papers out of them but also taught us grammar. I shall always remember your telling me never to split an infinitive. . . .

. . . I hope one day to bring my son to the E. T. Bell Museum of Pathologic Anatomy at the University of Minnesota and to say, "Ambrose, look at that name well, for Dr. Bell played a big part in your father's life. He taught me a lot of things about pathology and about the art of living which are not in books." . . .

Harry A. Wilmer, Rochester, Minnesota: . . . The ever-open door to your office, the ever-ready kindness with which you welcomed each intrusion into your crowded working day, and the continual production of significant works from your pen are the things which I remember particularly. I remember, also, your refusal to put your name as co-author on papers which I published, in which you were a collaborator of such importance that my name probably should have been second.

I suppose I summed up my feelings in the dedication of my Doctor of Philosophy thesis (1944) . . . As a medical student or fellow I was always treated as a colleague. Like all true debts of gratitude of the work-

men of science to their teachers, mine is so intangible as to be measureless and so singularly precious as to be above value. . . .

Raymond E. Buirge, Minneapolis: . . . A few years ago, while I was at Memorial Hospital in New York, I enjoyed the rare opportunity of a congenial visit with Dr. Ewing. After the mention of Minnesota, Dr. Ewing wanted to know if I knew you. To make a long story short, Dr. Ewing said, "I believe that Dr. Bell is the greatest living clinical pathologist in the world today."

Doctor Bell has received many honors. An example is the 1956 Distinguished Service Award of the Minnesota Chapter of the Society of the Sigma Xi, and the citation reads:

To Elexious Thompson Bell, Professor of Pathology Emeritus, School of Medicine, University of Minnesota, Dean of Pathologists in the Northwest, beloved friend of thousands of physicians and world renowned scientist. For almost fifty years, as teacher and author, you have enriched the lives of your students and colleagues; as a pathologist, you have raised the standards of medical practice; and, as a scientist, you have increased our understanding of disease. Your investigations of the kidney, culminating in the monograph, *Renal Diseases*, and your studies of hypertension, diabetes, and arteriosclerosis are outstanding examples of your contribution to medical science. Always unselfish and sympathetic, you have given counsel and courage to the troubled, and, by your example, you have influenced many to devote their lives to teaching, to service, and to research.

A more recent example is his honorary degree, D.Sc., from the University of Missouri, June 6, 1959:

Vobis hoc notum sit Praesidem Universitatis Reipublice Missouriensis Curatorum auctoritate ELEXIOUM THOMPSON BELL adornavisse atque auxisse eique fructu de disce omnia jura, honores, insignia quae ad gradum SCIENTIAE DOCTORIS evectis concedi soleant.

We salute you, Dr. Bell. Many of us are glad we know you affectionately as "Tommy." We wish we might have known you when you were a boy who went barefoot and rode horses bareback, and when you were a student at "Misson," and when you sampled "gutes Bier" at that pension in Bonn. We propose a toast to your health, highly esteemed friend, and to your continued productivity. *Auf Euer Wohl, hochgeschätzter Freund, auf weitere Jahre gesunder Schaffenskraft!*

SOME OF DR. BELL'S HONORS

- 1933—President, American Association of Pathologists and Bacteriologists.
- 1931—President, American Association for Cancer Research.
- 1939—Ludvig Hektoen Lecturer, Frank Billings Foundation, University of Chicago.
- 1910—Secretary, Minnesota Pathological Society (for a total of 29 years).
- 1946—Honorary member, St. Paul Surgical Society.
- 1948—Presidential Award, International Poliomyelitis Congress.
- 1949—Distinguished Service Award, Minnesota State Medical Association.
- 1944—Cap and Gown Day Lecturer, University of Minnesota.
- 1949—Honorary member, Dallas Southern Clinical Society.
- 1949—E. T. Bell Fund, Minnesota Medical Foundation, for the E. T. Bell Pathology Museum.
- 1951—Medal, American Cancer Society, Inc.
- 1952—Honorary member, Tacoma Academy of Medicine.
- 1952—Intern-Resident Lecturer, Mount Carmel Mercy Hospital, Detroit, Michigan.

- 1955—Announcement of E. T. Bell Lectureship, Minnesota Pathological Society.
- 1955—Unveiling of the E. T. Bell portrait, Minnesota Pathological Society.
- 1956—Distinguished Service Award, Minnesota Chapter of the Society of Sigma Xi.
- 1958—Shield of the Michigan Pathological Society (awarded to E. T. Bell, M.D., Carl Vernon Weller Lecturer).
- 1958—Membership, Fifty-Year Club, Minnesota State Medical Association.
- 1959—Medal, Minnesota Academy of Medicine.
- 1959—Banting Distinguished Service Medal, American Diabetes Association.
- 1959—D.Sc., honorary degree, University of Missouri.

CHRONOLOGIC LIST OF DR. BELL'S PUBLICATIONS

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2. Experimentelle Untersuchung über die Entwicklung des Auges bei Froschembryonen. *Arch. f. mikr. Anat.* 68:279, 1906.
3. Experimental studies on development of the eye and nasal cavities in frog embryos. *Anat. Anz.* 29:185, 1906.
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7. Staining of fats, in epithelium and muscle fibers. *Anat. Rec.* 4:199, 1910.
8. Interstitial granules of striated muscle and their relation to nutrition. *Internat. Monatschr. f. Anat. u. Physiol.* 28:297, 1911.
9. Interstitial granules (liposomes) in fatty metamorphosis of striated muscle. *J. Path. & Bact.* 17:147, 1912.
10. Cloudy swelling: a preliminary report. *J.A.M.A.* 61:455, 1913.
11. Study of the lesions produced by *Bacillus proteus* (with W. P. LARSON). *J. Infect. Dis.* 13:510, 1913.
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16. Renal tumor of the rabbit (with A. T. HENRICH). *J. Cancer Res.* 1:108, 1916.
17. Renal tumor in the rabbit (with A. T. HENRICH). *J. Cancer Res.* 1:157, 1916.
18. Tumors of the thymus in myasthenia gravis. *J. Nerv. & Ment. Dis.* 45:130, 1917.
19. Pathology of the lungs in pneumonia following influenza. *Journal-Lancet* 39:3, 1919.
20. Perfusion experiment in study of cellular anaphylaxis (with W. P. LARSON). *J. Nat. Dent. A.* 6:453, 1919.
21. Effect of foreign protein on the kidney (with T. B. HARTZELL). *J. Infect. Dis.* 24:618, 1919.
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24. Infantile spinal progressive muscular atrophy (Werdnig-Hoffmann): report of a case with necropsy findings (with E. J. HUENEKENS). *Am. J. Dis. Child.* 20:496, 1920.
25. Etiology and development of glomerulonephritis (with T. B. HARTZELL). *Arch. Int. Med.* 29:768, 1922.
26. Studies on hypertension: relation of age to size of heart (with T. B. HARTZELL). *J. Med. Res.* 44:473, 1924.
27. Outline of Pathology. Minneapolis: Univ. Minn. Press, 1924, 586 pp.
28. Experimental glomerulonephritis (with B. J. CLAWSON and T. B. HARTZELL). *Am. J. Path.* 1:247, 1925.
29. Distinction between chronic glomerulonephritis and hyperpiesia (primary hypertension). *Minnesota Med.* 9:1, 1925.
30. Comparison of acute rheumatic and subacute bacterial endocarditis (with B. J. CLAWSON). *Arch. Int. Med.* 37:66, 1926.
31. Valvular diseases of the heart, with special reference to pathogenesis of valvular defects (with B. J. CLAWSON and T. B. HARTZELL). *Am. J. Path.* 2:193, 1926.
32. Circumstances that influence the obtaining of necropsies. *J.A.M.A.* 90:896, 1928.
33. Primary essential hypertension: a study of 420 cases (with B. J. CLAWSON). *Arch. Path.* 5:939, 1928.
34. Lipoid nephrosis. *Am. J. Path.* 5:587, 1929.
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39. Renal lesions in toxemia of pregnancy. *Am. J. Path.* 8:1, 1932.
40. Relation of lipid nephrosis to nephritis. *Ann. Int. Med.* 6:167, 1932.
41. Glomerular lesions associated with endocarditis. *Am. J. Path.* 8:639, 1932.
42. Amyloid disease of the kidneys. *Am. J. Path.* 9:185, 1932.
43. Renal lesions associated with multiple myeloma. *Am. J. Path.* 9:393, 1933.
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56. Primary hypertension: clinical and pathological studies of 1,520 cases with special reference to renal arteriosclerosis. *Proc. A. Life Insur. Med. Dir. America* 26:269, 1940.
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61. Tumors of the breast. *Texas State J. Med.* 38:537, 1943.
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64. Hyperplasia of pulmonary alveolar epithelium in disease. *Am. J. Path.* 19:901, 1943.
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- strain of mice (with A. KIRSCHBAUM and J. GORDON). J. Lab. & Clin. Med. 34:209, 1949.
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89. Renal vascular disease in diabetes mellitus. Diabetes 2:376, 1953.
90. Decline in mortality from syphilis in Minnesota. Arch. Path. 59:259, 1955.
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97. Pancreatitis. Surgery 43:527, 1958.
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INTRAVENOUS administration of streptokinase can cause artificially induced intravascular clots to dissolve. Induction of thrombi can be affected by direct irritation of the intima with a dental broach or by chemical irritation with sodium morrhuate. The position and size of the thrombi can be studied by clinical observation and venograms.

The plasmin system, believed to be responsible for physiologic lysis of clots, is activated by intravenous infusion of purified streptokinase, which reacts with a proenzyme in plasma to form an activator complex. This, in turn, reacts with plasminogen to form the proteolytic enzyme, plasmin. Inhibition of this complex system is possible at 3 levels: neutralization of streptokinase by specific antibody; neutralization of the activator complex by an inhibitor in plasma; or neutralization of plasma by an inhibitor.

Administration of an initial priming dose of streptokinase is essential in order to neutralize any circulating antibody and streptokinase inhibitor. The priming dose varies from approximately 83,000 to 682,500 units, the exact amount being calculated by quantitative determination of the antibody and inhibitor in the blood.

Clots more than 24 hours old are resistant to lysis regardless of the method employed.

A. J. JOHNSON and R. McCARTY: The lysis of artificially induced intravascular clots in man by intravenous infusions of streptokinase. J. Clin. Invest. 38:1627-1643, 1959.

Book Reviews

A Synopsis of Anesthesia

J. ALFRED LEE, M.R.C.S., 1959. *Baltimore: Williams & Wilkins Co.* 624 pages. Illustrated. \$6.50.

As stated in the preface, this text is designed to serve as a summary of current teaching and practice and as a source of reference. The author hopes that the material will stimulate additional reading from original sources.

There is a wealth of material available in this relatively small text (573 pages). The student of anesthesia (especially at the elementary level), the part-time practitioner, and those participating in dressing room debates will find this text to be a source of preliminary answers to questions. As the author has indicated, it is not intended to be a complete text in the field.

I feel, however, that the book suffers from several major defects. One of these is the rather peculiar organization of the material. Why is the regulation of heart action included under "Adrenergic Drugs" rather than under the "Physiology of Circulation?" Why is the discussion of posture included under "Pneumothorax Preparation?" Why are cyclopropane and nitrous oxide discussed separately (cyclopropane under "The Closed System") rather than included in the section on "Agents for Inhalation Anesthesia?" Finally, the discussion of inhalation anesthetic systems is mixed up with the discussions of cyclopropane and nitrous oxide. One of the major advantages of a summary type text is the ready accessibility of answers to specific items, and the disorganization of the material detracts from this function.

Since in the United Kingdom, Europe, and the United States drugs have different trade names, the search for material on a specific drug would be facilitated if generic terms were used rather than trade names.

I am concerned also about the relative space allotted to various items; for example, much more space is given to nitrous oxide than to other inhalation agents. This may represent a special interest of the author or the perpetuation of a balance of importance that once existed.

In some sections there seems to be a dominance of basic anatomy (material which can be secured as easily from standard anatomy texts) as compared to function. For example, a limited discussion of maintenance of blood pressure follows an extensive outline of the anatomy of the nervous system.

These are other independent items I question. Why are there no important dates after 1950 in a 1959 edition? There seems to be no mention of alveolar ventilation. Reactions to depressant drugs such as opiates and barbiturates are not outlined, and the antinarcotic drugs are mentioned in a separate chapter. There is an extensive chapter on regional anesthesia (without many associated illustrations); I question the propriety of this chapter in a text designed as a summary. No discussion of flow rates and their impact upon different anesthetic systems is included. There is discussion of and pictures of mouth props and gags—most modern anesthetists have abandoned these devices.

Because much material has been included that could have been excluded in a text designed to function as an outline or a summary, it has been necessary to use small type to effect a small book. The small type makes reading more difficult.

STUART C. CULLEN, M.D.
San Francisco

Tuberculosis and Other Communicable Diseases

J. ARTHUR MYERS, M.D., Editor, 1959. *Springfield, Ill.: Charles C Thomas.* 499 pages. Illustrated. \$14.50.

The inclusion of other important infectious diseases with tuberculosis serves the very useful purpose of placing common emphasis upon etiology and communicability. This emphasis may be the key that will help unlock effective preventive measures. While this volume has public health emphasis, it contains excellent clinical information and will be a valuable addition to the working tools of the internist, general practitioner, and student of medicine or nursing.

The chapter by Dr. Myers is a clear interpretation of primary (first infection) tuberculosis with its excellent prognosis. This contrasts with the reinfection (endogenous) or clinical type which may be acute or chronic. From broad experience, Dr. Myers notes that BCG has shown no advantage over the conservative measures as often used in the United States. The extent of incidence of tuberculosis is shown by the fact that about one-third of the population of the United States reacts positively to tuberculin testing.

In subsequent chapters, 21 respiratory diseases, including influenza, are discussed. Here emphasis is upon etiology. It is recognized that the clinical spectrum represented by these diseases is broad, indeed, ranging from the mildest manifestations to very marked variations. Of 21 acute infectious diseases of respiratory nature, 12 were ascribed as due to viruses. It is seen that the adenoviruses have become important pathogens. Extensive epidemics of pharyngoconjunctival fever have been caused by such pathogens.

Current additions to the literature are included in the bibliography. For instance, the information with regard to Coxsackie, Echo, adeno- and other viruses are brought up to date. This valuable text emphasizes the importance of the viral infections.

C. A. MCKINLAY, M.D.
Minneapolis

Psychiatry in General Practice

J. A. WEIJEL, M.D., 1959. *Houston: Elsevier Press, Inc.* 208 pages. \$7.00.

This is a compact volume which presents the thinking and experience of the psychiatrist member of a productive group of Dutch workers in the field of social medicine. The book is a unique mixture of practical instructions for arriving at an understanding of the psychologic and social components to disease in distress (in the form of a psychosocial questionnaire); reflections on some of the subtle, conceptual, and philosophical problems which underlie the almost universal difficulties which the medical profession has with this part of its job; and a more conventional description of psychotherapy with its instructions for its application.

The author begins by pointing out that, in general, psychiatry has been used in consultation in the same way as the other medical specialties, namely, to determine whether or not a disease exists in the area of that particular specialty. There is, however, a difference between psychiatry and specialties such as otology and ophthalmology.

(Continued on next page)

BOOK REVIEWS

(Continued from page 45)

mology, namely, that the "psyche" cannot be considered an organ, which makes this kind of use inappropriate. Dr. Weijel further states that the knowledge and methods of psychiatry as a specialty are not directly applicable to general practice and have not been made available for practicing doctors in a form which has been useful.

The fact is emphasized that, in medical practice, the doctor's naïve acceptance of the patient's somatic complaints at face value sets the "whole medical apparatus blindly in motion." He makes a plea for the consideration of psychosocial factors along with the organic ones at the outset of the medical contact and offers the tool of his own devising, the more than 90 questions of the Psychosocial Questionnaire, as a way of doing this. He states that "the real medical question is, Why does a patient complain, not what disease does the patient have."

Another section of the book which is hard going, but which deals incisively with a difficult problem, is that wherein Dr. Weijel considers the dilemma between treating the patient objectively as a "case" of, for example, diabetes or treating him as "a human being," that is, treating his subjective feelings. Both of these points of view are useful and meaningful. Dr. Weijel states that "Both attitudes are exaggerations of the same reality and are always correct up to a certain degree."

The Psychosocial Questionnaire is a disarmingly simple series of questions, but the author's description of its use and clinical examples explains its subtlety and depth. It is brief enough to be incorporated as a part of any careful medical history. I have used this questionnaire and find it a useful adjunct to the spontaneous recital of symptoms obtained by a nondirective type of interviewing—what Deutsch has described as "associative anamnesis."

The last section of the book is concerned with the more conventional description of psychiatric syndromes and clarification of the application of psychotherapy in general practice. Particularly noteworthy is Chapter 13, "The Importance of Diagnosis."

In summary, while this is not a textbook, there are sections of it which can be put to immediate use by every practicing physician. It contains an easily digested, very useful diagnostic tool, the "Psychosocial Questionnaire."

The author's philosophic considerations are profound and will be harder to grasp. However, they involve the vital issues in medical practice today. They should be considered by all physicians who are concerned about the impact of specialization and expanding knowledge on medicine and the apparent decline of medicine's prestige and its transition to a technical rather than a service profession.

RICHARD NIAGRAW, M.D.
Minneapolis

Textbook of Pediatrics

WALDO E. NELSON, M.D., Editor, 1959. Philadelphia: W. B. Saunders Co. 1,462 pages. Illustrated. \$16.50.

Textbook of Pediatrics, seventh edition, formerly Mitchell-Nelson Textbook of Pediatrics, is an excellent reference. Since this edition is the most recently published of any of the general standard pediatric texts, it can be expected to be the most up to date with the rapid expansion of medical knowledge in this field.

One will note the list of contributors, 81 in all, each an authority on the particular field on which he writes.

Most of these are the same authors or their collaborators with carefully worked out revisions from previous editions, but there are many new contributors who have produced completely new sections, such as those on parenteral fluid therapy, drug therapy, and rickettsial diseases. There are additional subjects not in the previous text, for example Kala-azar tropical eosinophilia, mesenchymal diseases, behavior problems associated with organic brain damage, and so on.

This book is both compact and complete, having some 200 fewer pages than previously, with greater variety of material, and without omitting any of the important aspects. Among the points which make it an outstanding reference for any physician who deals with children are the very fine coverage of normal growth and development through adolescence, including graphic tables, and the excellent scientific and sensible exposition of normal mental and emotional development, along with the more commonly encountered behavior disorders from infancy to maturity.

To be mentioned also, as of special value to the practitioner, is the table of drugs giving in graphic form the dosages and available preparations of most of the medications in present day usage and methods of figuring dosage by weight and surface area. The appendix includes tables of normal values for blood and spinal fluid constituents; conversion tables for milligrams to milliequivalents, apothecary and metric equivalents, and Fahrenheit to centigrade temperature; and a good table of food values, diet calculations and food exchanges. The chapter on parenteral fluid therapy will be found most useful because of the clarity of clinical exposition of disorders and needs and the practical outline of type of fluids required, with the available preparations.

The above mentioned are only examples of the help and information to be gained by reference to this book. All phases of childhood and adolescence relative to health and disease are well covered, as is expected in a complete textbook. Most impressive is the accuracy and detail in presentation of material, which results from the fundamental knowledge of the most recent developments in pediatrics.

ELIZABETH C. LOWRY, M.D.
Minneapolis

The Care of Minor Hand Injuries

ADRIAN E. FLATT, M.D., 1959. St. Louis: C. V. Mosby Co. 257 pages. Illustrated. \$9.50.

The busy clinician is always pleased when a book is produced that is practical, well illustrated, and easily read and treats adequately the included subjects. Adrian E. Flatt, author of "The Care of Minor Hand Injuries," and The C. V. Mosby Company have accomplished this mission.

Properly trained physicians realize that many pathologic conditions of the hands are not of minor character. However, the author has exercised his privilege and selected the above title, "the word *minor* implying a relatively small amount of tissue damage, outpatient treatment, and freedom from postoperative complications." In a number of large teaching hospitals, some of the clinical entities of the hand which are discussed in this publication would be treated in the hospital at first and subsequently on an ambulatory basis. Past experience has resulted in a policy of serious consideration and respect of patients who have sustained trauma to the hands.

The contents of this excellent monograph are divided into 2 main sections, the first of which is aptly entitled

(Continued on page 26A)

relieves both pain and stiffness with speed and safety

"... SOMA is very effective in decreasing paravertebral muscle spasm and the associated back pain. Its administration is simple; it is effective for reasonably long periods of time; and evidences of toxicity are rare even on prolonged use."²²

RESULTS WITH SOMA IN THE LOW BACK SYNDROME

Excellent to very good 68%

Good to fair 23.7%

Investigators' reports to the Medical Department, Wallace Laboratories. (Total of 278 cases)

NOTABLE SAFETY—extremely low toxicity; no known contraindications; side effects are rare; drowsiness may occur, usually at higher dosage

FAST ACTION—starts to act promptly **SUSTAINED EFFECT**—relief lasts up to 6 hours

EASY TO USE—usual adult dose is one 350 mg. tablet 3 times daily and at bedtime

SUPPLIED—as white, coated 350 mg. tablets, bottles of 50. Also available for pediatric use: 250 mg. orange capsules, bottles of 50

SOMA

T. M.
(carisoprodol Wallace)

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Literature and samples on request

WALLACE LABORATORIES, New Brunswick, New Jersey



BOOK REVIEWS

(Continued from page 46)

"General Principles of Care." The subject of the introductory chapter is functional anatomy. This unit is easily read, and the illustrations are exceptionally clear and helpful. One of the outstanding chapters concerns principles of care. A large number of topics are presented adequately, including anesthesia, the control of infection, physiotherapy, and disability among others. Some of the various points in surgical technic are described, and an interesting and definitive classification of wounds is presented with details concerning their clinical examination.

The remainder of the book concerns the care of specific injuries. Wounds of the skin and nails and pulp loss are explained with their management. Additional well-written chapters concern amputations, crush injuries, fractures, burns, infections, injuries of tendons and joints, and miscellaneous injuries and wounds.

This book was "planned to help those members of the profession who may be forced by circumstances to treat these cases without the help of previous experience." In the preface, the author also states that some of his colleagues may not approve of the inclusion of certain procedures. This is true, for some specialists in hand surgery will take exception to the use of thenar flap operations in a working man's hand. Nevertheless, the purpose of a medical monograph is to inform and instruct the profession. This has been well done by the author and the publisher.

EARL B. SANBORN, M.D.
Evanston, Illinois

Cerebral Angiography in the Management of Head Trauma

CHARLES A. CARTON, 1959. Springfield, Ill.: Charles C Thomas, 143 pages. \$7.00.

This volume is a monograph of the American Lectures in Surgery. It is divided into an extensive review of the literature and the review of 1,000 cases of acute and chronic injuries to the head for which 103 patients underwent angiography. One hundred eleven angiograms were made among 103 patients, and the angiograms of 51 patients demonstrated surgical lesions. The angiograms were made with the aid of local anesthesia, and the contrast medium was 35 per cent iodopyracet (diodrast), mrokon sodium, or thorium dioxide (thoratrast). Angiograms were made among patients who exhibited deterioration of the central nervous system or those whose general condition did not show any improvement. Thirty-six patients were found to have subdural hematomas, either acute or chronic. Acute subdural hematomas were found in the lateral positions over the hemispheres of the brain, whereas the situation of the chronic lesions was more nearly superior, over the hemispheres. The author believes that the use of tangential views in the angiograms is very helpful in localizing a subdural hematoma. One of the most prominent findings was a shift of the vessels away from the skull, with evidence of shift of normal venous sinuses. Bilateral subdural hematomas were likely to show displacement of the middle and posterior cerebral arterial systems but did not show as much shift of the anterior cerebral arteries. Nine epidural hematomas were found; those situated over the convexity of the brain produced displacement of the arteries, whereas epidural hematomas situated more basilar showed a lifting of the vessels. The most pathognomonic finding in angiograms of patients with epidural hematomas was a shift of the venous sinuses

away from the skull. This shift of the venous sinuses was not present in patients with subdural hematomas. Often a ragged cortical line was found suggesting an adherent dural clot.

The author believes that burr holes were most nearly accurately placed for subdural and epidural hematomas after angiography. Eighteen patients with intracerebral hematomas were included in this study, and 12 of these patients had other lesions; 7 had subdural hematomas and 1 had an epidural hematoma. It is also emphasized that, when a patient gives a history of injury to the head and angiography suggests hydrocephalus, the possible presence of a hematoma of a posterior fossa or some other lesion must be considered very seriously. Several patients who had depressed fractures, gunshot wounds, and cortical lacerations underwent angiography. It was found that angiography could give evidence of the extent of the bleeding and of the encroachment of bone upon the dura and substance of the brain.

It is interesting to notice that in this series the trauma was superimposed upon other lesions, such as 4 aneurysms, 1 angioma, and 1 arteriovenous anomaly. Three deaths directly attributable to angiography occurred. Results of angiography were falsely positive in only 2 patients, whereas in 11 some angiograms interpreted as indicating no abnormality actually, upon further study, revealed the patient to have an inoperable intracranial lesion.

The author has compiled an interesting evaluation of the deaths and complications in this series, and each case has a short summary in a very comprehensive table. The author also concludes that the information derived from the use of cerebral angiography in acute trauma to the head far surpasses the risk of the procedure and that the use of this method of diagnosis enables one to treat head injuries in a more intelligent manner.

ROSS H. MILLER, M.D.
Rochester, Minnesota

The Preparation of Medical Literature

LOUISE MONTGOMERY CROSS, M.A., 1959. Philadelphia: J. B. Lippincott Co. 361 pages. Illustrated. \$10.00.

The purpose of this book, as stated in the preface, is to present in convenient form the technics of preparing medical literature for publication in a book or journal article. The book describes in detail planning the paper or book, gathering the material, the actual writing, style, illustrations, and editing. Two useful appendices list annual publications, yearbooks, and reviews and the names and abbreviations for all journals regularly indexed in this country.

The author gives specific examples of good and bad titles, paragraphs, sentences, and choices of words. These are excellent and should be of great help to the novice. Her suggestions for writing a summary and conclusions, if heeded, go a long way toward eliminating the uninformative summaries that all too often find their way into print.

The author was perhaps ill-advised in going into rules of style because these vary so greatly from one editorial office to another that final styling must be left up to the publisher's copy editors. Her descriptions of printers' marks are a bit laborious, and her purpose could perhaps have been better served by showing examples. Despite these minor criticisms, this book is highly recommended for its thoroughness and the soundness of its approach.

WAYNE G. BRANDSTADT, M.D.
Chicago

(Continued on page 29A)

BOOK REVIEWS

(Continued from page 26A)

A Manual of Anaesthetic Techniques

WILLIAM J. PRYOR, M.D., 1959. Baltimore: Williams & Wilkins Co. 228 pages. Illustrated. \$7.00.

The rapid and continued progress of anesthesia necessitates frequent appraisals of newly introduced agents, apparatus, technics, and treatment of anesthetic complications. Most of these appraisals appear in the form of a review, monograph, or text covering one or more contributions. Recently, "A Manual of Anaesthetic Techniques" by Dr. William J. Pryor, senior anesthetist at The London Hospital, was published, representing a short, concise reference book especially prepared for house surgeons or registrars (residents) in anesthesia. Emphasis is entirely on recommended anesthetic techniques, based on recent literature and the author's experiences and presented with a minimum of theory.

The monograph is compact and excellently prepared. It is well organized, comprehensive, beautifully illustrated with diagrams and photographs, and well indexed. Containing many references, most of which are from British medical journals, the book is well printed on excellent paper. It is apparently designed, however, for anesthesiologists in Great Britain, since many of the apparatuses and technics are employed there and not in the United States. There are also certain objections, based on the technics used in America, to the procedures recommended by the author, for example, the use of a 16 gauge needle for a cricothyroid puncture (transtracheal anesthesia), administration of atropine gr. 1/150 to infants up to 1 year of age as premedication, use of thio-

pentane-ethylchloride-ether sequence, use of a 9 liter 500 cc. (N₂O-O₂) concentration for induction, and the use of general anesthesia in the majority of cases before administration of spinal anesthesia. Certain statements made by the author might be seriously questioned by the American anesthesiologist, such as "Trilene is not toxic to the liver and kidneys;" "Succinylcholine does not cross the placental barrier;" and "A tinge of cyanosis indicates that a patient is properly anesthetized with nitrous oxide oxygen."

STEVENS J. MARTIN, M.D.
Hartford, Connecticut

Synopsis of Treatment of Anorectal Diseases

STUART T. ROSS, M.D., 1959. St. Louis: C. V. Mosby Co. 240 pages. \$6.50.

Although identified as a synopsis of treatment, this book also includes brief diagnostic descriptions and pictures of disease processes of the colon and rectum. As a small book devoted to proctology, it is superior to any comparable outline, chiefly due to the step-by-step illustrations and photographs which clearly convey the treatment recommended by Dr. Ross. Not only is the treatment of common anorectal conditions described but there are also short chapters devoted to colonic malignancy, ulcerative colitis, and less common diseases such as hidradenitis suppurativa and sacrococcygeal cysts and tumors. As a brief, to-the-point, ready reference book, it is an excellent addition to any student's or practitioner's library.

WILLIAM T. SMITH, M.D.
Minneapolis

(Continued on page 30A)

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BOOK REVIEWS

(Continued from page 29A)

Synopsis of Ophthalmology

WILLIAM H. HAVENER, M.D., 1959. St. Louis: C. V. Mosby Co. 288 pages. Illustrated. \$6.75.

Dr. Havener, who is chairman of the department of ophthalmology at Ohio State University, has prepared with unusual insight a compact volume for the family physician that contains the ophthalmologic information that he needs and can use. The following are among the points accentuated.

The most rewarding single test of ocular function is the evaluation of visual acuity, and a test with the Snellen chart should be a part of every complete physical examination. To secure adequate mydriasis for ophthalmoscopy, neosynephrine, 10 per cent, is recommended. The routine use of the tonometer will detect unsuspected glaucoma in 2 per cent of patients over 40. An early diagnosis will prevent reduced acuity, field loss, cupped disk, and blindness. The most effective emergency treatment for chemical burns of the eye is immediate irrigation with tap water continued for fifteen minutes or more to get rid of every trace of the noxious agent. Injuries of the eye require tender and thoughtful handling. The eyeball should be carefully inspected before a lid laceration is sutured. For the removal of a foreign body embedded in the cornea, a sterile hypodermic needle of 20 gauge is suitable. (The needle should not be used for any other purpose.) Anesthetic eye drops or ointments should never be prescribed for home use, as they interfere with corneal healing. The differential diagnosis of the red eye is all important, since blindness can result from the wrong treatment. Steroid therapy must be used with discrimination, as it is known to aggravate infections, particularly herpetic keratitis. Exophthalmos of the thyrotoxic and thyrotrophic types is differentiated. Strabismus cases must be referred early for suppression amblyopia to be adequately treated. If symptoms of congenital impatency of the naso-lacrimal duct persist after the infant is 3 months old, probing is indicated.

Throughout the book, the writing is just right for its purpose—neither too heavy nor too light. The book is lucid, authoritative, adequately illustrated, and quite up to date. The general practitioner should find it of great service.

JAMES E. LEBENSOHN
Chicago

The Anastomoses Between the Leptomeningeal Arteries of the Brain

H. M. VANDER ECKEN, M.D., 1959. Springfield, Ill.: Charles C Thomas. 176 pages. \$7.50.

This monograph is the English translation of a thesis submitted by Dr. Vander Ecken to the University of Ghent. It is actually an enlargement of a publication by Vander Ecken and Adams in the American medical literature of 1953. The basic information in this monograph is, therefore, a repetition of the earlier publication.

The author studied the cerebral circulation in detail in many specimens, using a corrosive technic with concentrated hydrochloric acid after injection of the arteries with synthetic latex. In various specimens he demonstrated leptomeningeal precapillary anastomoses between all the major cerebral arteries and also between the leptomeningeal arteries of the cerebellum. There was con-

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BOOK REVIEWS

(Continued from page 30A)

siderable variation among individuals, and even between the two hemispheres of the same individual. He was unable to demonstrate anastomoses of significant size in the deep perforating arterics of the brain.

There is an excellent account of the author's observations on fetal leptomeningeal arteries from stages of development as early as 74 days to the neonatal period. By means of these observations, he develops chronologically the pattern of arterial development, including the origin of the leptomeningeal anastomoses between branches of the major cerebral arteries. Even though one may not readily accept the postulates concerning the causal relationships of the normal regression of the anastomotic channels in late fetal life, rejection of the postulates does not detract from the important chronologic observations made.

Clinicopathologic observations support the idea that these leptomeningeal arterial anastomoses play an important role in limiting the total area of some cerebral infarcts when a major cerebral artery is occluded. This is not so in regard to the deep perforating arteries, which do not have these anastomoses.

For the scholar interested in details of an anatomic-pathologic investigative technic, this monograph is worthwhile. The excellent review of the earlier literature building up to these data and the many photographs demonstrating the points made in the text contribute further to making this volume a worthy contribution.

JACK P. WHISNANT, M.D.
Rochester, Minnesota

The Mediastinum

TED F. LEIGH, M.D., and H. STEPHEN WEENS, M.D.,
1959. Springfield, Ill.: Charles C Thomas. 240 pages.
Illustrated. \$11.50.

When one considers the importance of many of the mediastinal structures, such as the heart, great vessels, trachea, or esophagus, and realizes that a pathologic process involving any one or all of them may seriously threaten a patient's life, the need for prompt and accurate diagnosis becomes readily apparent. This need has become even more apparent in recent years with the great advances in thoracic surgery, radiotherapy, and medicine in general, since many mediastinal conditions now have become amenable to therapy. For several years, it has been realized that no American text was available dealing with the radiologic diagnosis of mediastinal lesions.

This comprehensive monograph by Drs. Leigh and Weens very nicely fills this gap in the radiologic literature and is probably the first of its kind in the English language devoted entirely to the radiologic aspects of the normal and abnormal mediastinum. The broad experience of the authors plus their extensive review of the related literature, including certain valuable articles from abroad, makes this an authoritative work.

The monograph contains 30 well-organized and carefully written chapters. Chapter 1 concisely covers the normal anatomy of the mediastinum. Of particular interest are brief discussions of how the classical anatomic descriptions based on cadavers are at variance with the true anatomy in the living patient observed radiologically. A separate chapter is devoted to a rather complete discussion of the anatomy of the mediastinal lymph nodes.

A typical chapter will deal with one related group of entities, such as neurogenic lesions, and briefly discuss for each entity the origin, incidence, size, location, clinical complaints and findings, differential diagnosis, related diseases, gross pathology, and, finally, radiologic findings. As one would expect, the radiologic findings are completely discussed along with any helpful distinguishing x-ray features, such as patterns of calcification or associated bony abnormalities, which might help establish a specific diagnosis.

Common as well as many of the rarer mediastinal lesions are covered, as illustrated by these selected chapter titles: "Diaphragmatic Hernias," "Thyroid Lesions," "Bronchogenic Cysts," "Mediastinitis," "Teratomas," "Pericardial Lesions," "Duplications of the Alimentary Tract," and "Extramedullary Hematopoiesis." One important mediastinal organ, the heart, has been deliberately excluded, since heart disease is a large subject in itself, adequately handled in existing texts. Although there is no chapter dealing with the various special technics used in the radiologic work-up of mediastinal lesions, these are adequately discussed throughout the book where applicable.

The monograph is profusely illustrated with good anatomic drawings and representative, well-reproduced radiographs. Adequate bibliographic data are conveniently arranged at the end of each topic, and the extensive cross index should prove helpful for easy localization of subject matter. Now it should be possible for the radiologist who is often confronted with an abnormal mediastinal contour to systematically obtain the proper additional radiologic studies. From these he can render an accurate differential diagnosis or may even make a specific diagnosis by referring to a single text instead of expending time and effort in searching for separate articles from many sources.

The book constitutes a long-needed and extremely useful contribution to the reference libraries of those physicians charged with the accurate diagnosis and treatment of mediastinal lesions, especially the radiologist, chest physician, thoracic surgeon, and pathologist.

DONALD B. ROONEY, M.D.
Emory University, Georgia

Molecules and Mental Health

FREDERIC A. GIBBS, M.D., Editor, 1959. Philadelphia:
J. B. Lippincott Co. 189 pages. \$4.75.

This volume brings together papers and their discussions presented at two conferences held in 1958 under the auspices of the Brain Research Foundation of Chicago. The first deals with "Amines in Relation to Brain Function and Behavior" and the second with "ACTH Treatment of Hypsarrhythmia." The section on amines gives reports on ceruloplasmin, taurine (Heath), tryptophan metabolism, ergot hallucinogens, epinephrine and norepinephrine, and acetylcholine as they may relate to behavior and mental illness. This is a pioneering and hence controversial region of research. These papers are of particular interest to investigators working along related lines.

The second half of the book, on "Hypsarrhythmia," is of special interest to practitioners and particularly to pediatricians, since it describes a little known neurologic condition of the first year of life which is said to have a favorable prognosis with ACTH therapy.

DONALD W. HASTINGS, M.D.
Minneapolis

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OPHTHALMOLOGY

*Introduction to a Series of Articles on Ophthalmology
and Related Subjects which may be of interest to the
busy family doctor.*

MALCOLM McCANNEL, M.D.

THE BUSY FAMILY DOCTOR encounters ocular disorders every day of his practice. He does not, and rightfully should not, try to handle all those he sees. He does, however, want to be able to diagnose and treat cases which rightfully fall within his purview.

In the best interest of his patients, he wishes to be aware of the newer developments in the field of ophthalmology and, at the same time, have a clear-cut idea of the prognosis and outlook for the more common problems that are encountered.

In this series of articles dealing with ophthalmology beginning in this issue of THE JOURNAL-LANCET, many areas will be explored and information given in the hope that the family physician will be able to diagnose earlier, cooperate in the management of the disorder, and often supervise part of the treatment intelligently as he continues his role as the family's medical confidant and adviser.

THE JOURNAL-LANCET would welcome suggestions from its readers on topics that may be missed or overlooked.

The first of these articles is written by Dr. Bourne Jerome, a clinical professor of ophthalmology at the University of Minnesota. Dr. Jerome's paper deals with vision, eyestrain, and glasses.

Vision, Eyestrain, and Glasses

BOURNE JEROME, M.D.

Minneapolis, Minnesota

THIS IS THE AGE of enlightenment, or of striving for it, in which our average citizen possesses more usable knowledge of the body and its ills than did the most learned physicians of Washington's time. Most readers will agree that this average citizen is happier and cares for family and self more intelligently for having this knowledge. Logically then, more enlightenment is worth working for, especially in fields in which our average citizen's concepts seem to remain comparatively faulty. The eye, we ophthalmologists think, is almost the prime example of such a field.

Patients expect their family medical adviser to be guide and interpreter in matters of the eye, a job he can do confidently if he's equipped with sound information to replace their legends and fallacies and to orient them to means of efficient and comfortable seeing. In the family physician's behalf, I'd like to discuss here some medical concepts and popular misconceptions about *vision*, *eyestrain*, and the *role of glasses* in correcting blurred and strained vision.

Vision tests, using the standard Snellen illiterate E and letter charts, are routine in the schools and are used more and more in doctors' offices. We hope that all of the schools in our area use Snellen charts rather than mechanical gadgets! With any method of determining visual acuity, the human element must be considered. The examiner will find variations in response such that two patients with precisely similar eyes can conceivably differ in their performance by one, possibly two, lines on the test chart. A tense patient may subconsciously overaccommodate and blur letters that he could readily read if put at his ease. Thus the figures obtained for the smallest print a patient reads cannot be

related with mathematical exactness to his real visual efficiency. School-sponsored vision testing is aimed at finding those students with acuity enough below 20/20 to be suspected of being handicapped. It is a screening process and may defeat its own purpose if too meticulously performed and interpreted.

A word is in order here concerning the meaning of the fraction 20/20. It's an arbitrary standard of normalcy based on the three-century-old observation of the astronomer Hooke that a good eye could, on a clear night, barely distinguish a certain star-pair as separate entities. The angle formed at the eye between rays from each star—one minute—is the angle formed by the thickness of the stroke forming a 20/20 letter, at one's eye, 20 ft. away. If one can just distinguish this size letter, his vision is 20/20; if he must approach to 15 ft. to identify it, he has 15/20 vision; if he can barely read a letter twice the size at 20 ft., his vision is 20/40.

Seeing is half physical and half mental. One who treats the eye must be guarded in accepting a small increment of test chart acuity as a valid sign of improvement in disease. Also, the patient who sees 20/30, takes eye exercises, and then sees 20/25, has perhaps improved *on the chart*, but research shows that he cannot carry over this improvement to, for example, more accurately identify a ship on the horizon than he could before exercising.

Eyestrain is a convenient term for an assortment of well-known symptoms resulting from frequent or difficult adjustments and readjustments of the neuromuscular mechanism in, on, or behind the eyes. These symptoms include aching, rapid fatigue, headaches, irritability from hyperemia of lids, and conjunctiva induced by overworking the eyes. Clinically, we know that intensified neuromuscular adjustments result from: (1) a difference between the eyes in respect

BOURNE JEROME is a clinical assistant professor of ophthalmology at the University of Minnesota.

to refractive power or size of retinal image; (2) a tendency to turn out of line with each other, requiring tonic effort to maintain parallelism; and (3) the rapid alternation of focus for first one meridian and then the other that characterizes low-grade astigmatism. High astigmatism can't thus be accommodated for; such a patient's eyes are comfortable but blurred. Reading by a dim light, poor print, or on a bus calls for rapid ocular readjustments to maintain fixation. Lancaster says, "Compare the effort required for holding the point of a needle or even of a pencil precisely over a required object point without anything on which to rest the hand. The muscular effort in foot-pounds or ergs is a trifling load for the muscles concerned, but the effort at precision and steadiness is exacting and very fatiguing."

It's tempting to believe that this eyestrain can bring about organic changes in the eyes, such as an increase in myopia or deterioration of retinal cell structure. Many people seem to believe this, but ophthalmologists have never found or had presented to them convincing evidence of such a cause and effect relationship. Most of them now believe that misuse of the eyes, even in childhood, by excessively prolonged reading, strained positions, or inadequate light, does not cause refractive errors to develop, intensify existing ones, or hasten the onset of degenerative ocular disease. Our ancestors, who read by firelight, had no worse eyes than we, and the eyes of aborigines, who can't read, seem, from all accounts, to fare no better than ours.

The physician who agrees that strained eyes will last just as long as unstrained eyes can render a real service by thus confidently reassuring the anxious patient whose eyestrain is unavoidable or whose guilt complex stems from assumed neglect of his own or his children's eyes.

Of course no sensible person will abide real discomfort if glasses abolish it or submit to a visual handicap if glasses can correct it. If his youngster's crossed eyes can be made straighter by glasses, he will enforce their use. If he himself works or plays where he's subject to intense light or flying particles, he'll wear protective goggles. We have thus just 4 basic indications for glasses: (1) ocular discomfort; (2) refractive error; (3) ocular incoordination; and (4) physical hazards. Misconceptions concerning all 4 abound and will be touched upon.

Glasses probably originated in China. Nero had reading glasses, but he must have removed them while watching Rome burn, for bifocals came along only after our own Revolution. Franklin, their inventor, did not live to see

cylindrical lenses used for correction of human astigmatism.

People have all sorts of notions about the need for glasses and what glasses will do for and to their eyes. The belief that prolonged or meticulous eye work requires glasses to aid even comfortable, clear-seeing eyes brings many a conscientious patient to the oculist. Rarely indeed does the latter find justification for prescribing lenses for such an individual or for the one who wants lenses prophylactically because lots of eye work looms ahead of him. The concept that wearing lenses may forestall future symptoms is simply not tenable. This relates to the usually mistaken belief cherished by some parents that if glasses are worn faithfully to correct subnormal vision or eyestrain during childhood, they will not necessarily be needed later on. The facts are: (1) hypermetropia only rarely decreases with growth to a point where the accommodative power is adequate to maintain clear, comfortable vision without aid from the previously essential lenses; (2) myopia tends to increase with growth and age, and, though glasses may be removed anytime without harming the eyes, except perhaps in the rare cases of high malignant myopia, distance vision will remain subnormal while they're off; and (3) astigmatism seldom decreases significantly with age, and a needed lens correction can seldom be successfully abandoned at any age.

Realizing that their newly myopic moppet must wear glasses into adulthood, parents may soften this impact by fondling another misconception—that changes in the eyes at middle age may render glasses for distance vision then unnecessary. Actually, the lessening of myopia due to tissue sclerosis in the 50's or later rarely suffices to allow much improvement in distance vision. Anyhow, this myopic moppet in his 50's will be more or less happily reconciled to bifocals. If he will successfully dodge this issue, he must remove his spectacles to read. He has always seen clearly at close range without them. Thus, while certain other advantages do accrue with maturity, the eyes are not then favored. The oldster, if he is nearsighted to the appropriate degree, can read fine print without glasses. This singular faculty is really the only consolation that myopia has ever offered him.

SUMMARY

This article deals with features of vision, eyestrain, and their relation to wearing glasses. Emphasis was placed on phases of these matters with which the family doctor is called upon to cope or advise. Vision testing must be conducted

with reasonable care, but normal variations in response preclude too strict interpretation of findings. The meaning of 20/20 vision was explained. Small improvements in acuity by exercise are more apparent than real. Eyestrain is not a result of too heavy a load on ocular muscles but of incessant readjustments by those muscles and their nerves. There is no good evi-

dence that eyestrain can change eyes organically and irreversibly, and worried patients should have this explained to them. Glasses aren't needed for comfortable and sharp-seeing eyes even for hard and detailed work. Except in strabismus, glasses never cure anything. They seldom are of value prophylactically and, if really needed early in life, tend to remain indispensable.

INTRINSIC TUMORS of the brain stem can most often be diagnosed as infiltrating gliomas. The majority of patients are under 20 years of age, and brain stem growths constitute approximately 15 to 20 per cent of all intracranial tumors in patients under 15. These tumors usually originate in and occupy the pons, but they may extend upward or downward from that location.

Paralysis of the external rectus muscles and of conjugate lateral muscles are among the commonest manifestations. Unless the tumor originates in the midbrain rather than the pons, the third cranial nerve is affected late, if at all.

The descending motor pathways and cerebellar connections are almost always affected, but sensory changes in the trunk and extremities are rare. Headache, nausea, and vomiting are common, and the patient may experience anorexia and difficulty in swallowing, eventually resulting in emaciation.

Most frequently, the sixth nerve will be affected early in the course of the disease. Impairment of the abducens nerve and consequent paralysis of the lateral rectus muscle produce double vision and convergent strabismus, obvious to the patient even when paresis is slight.

Lateral conjugate deviation of the eyes to the side of the tumor may be abolished, or movement of both eyes in a horizontal plane may be affected, probably due to interference with the connections between the abducens nucleus and the oculomotor nucleus through the median longitudinal fasciculus. The oculomotor nucleus innervates the contralateral internal rectus muscle, which is responsible for the lateral deviation of the eye opposite the paralyzed abducens nerve.

Probably the most characteristic manifestation of a pontine tumor is impairment of lateral rotation of the eyes. This symptom, when seen in a child together with abducens paralysis without increased intracranial pressure, strongly suggests the presence of a pontine tumor. Such paralysis appears rarely in cases of cerebellar tumors but may occasionally be associated with vascular lesions of the brain stem.

Acute disorders of the frontal lobes may also cause transitory disturbances of conjugate lateral movement, although these phenomena are sudden and of short duration. Gliomas occurring in the pons affect the facial nerve almost as frequently as the sixth nerve.

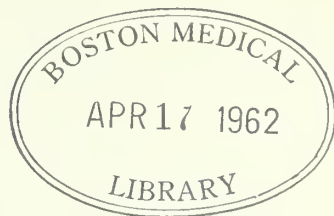
Nystagmus, either horizontal or vertical, is commonly associated with pontine tumors and is thought to be caused by damage to the vestibular nuclei.

P. C. BUCK and J. E. KEPLINGER: Tumors of the brain stem with special reference to ocular manifestations. *Arch. Ophthalm.* 62:541-554, 1959.

Hip Synovitis in Children

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THE LIMPING CHILD requires a careful diagnostic work-up (table 1). He may have poliomyelitis or Guillain-Barré acute polyneuritis, in which case signs or symptoms of central or peripheral nervous system disease are usually found. Facial weakness and an elevated spinal fluid protein with normal cell count favors Guillain-Barré syndrome. If this child has acute rheumatic fever, expected findings would be elevated temperature; swollen, hot joints; increased sedimentation rate; heart murmur; and evidence of previous streptococcal disease. Perthes' disease usually occurs in boys from 3 to 12 years of age. Slipping of the femoral epiphysis is most likely to be found in boys aged 10 to 16. Leukemia or neuroblastoma may start with bone pain and a limp. Sickle-cell disease may present with bone pain, fever, heart murmurs, and anemia and may mimic rheumatic fever.

The most common cause of a limp in childhood is hip synovitis. Of 139 patients with orthopedic disorders seen in my office, 118 were children with hip synovitis (table 2). I have collected 198 cases of hip synovitis in children from the literature¹⁻⁵ (table 3). This disease occurs predominantly in males at an average age of 6 years. It is also known as coxitis, arthritis, epiphysitis, and hydrarthrosis.

The child with hip synovitis awakens in the morning with pain in the hip or knee and is not able to walk without a limp. There is no history of trauma. He has no fever. There are usually no allergic manifestations present. In my own series of 400 allergic children, hip synovitis never developed in any of them. The child usually walks with the leg abducted and with the knee stiff. Hip motion is limited, but knee motion is normal. No swelling is seen. The joint may be tender over the anterior hip, and there is usually pain on abduction. The reflexes are equally

present in both legs. Most of these children have hypertrophied tonsils or chronic tonsillitis. Roentgenograms may show edema of the joint. There is apparently no relationship between this disease and osteochondritis. In none of the children from this clinic with hip synovitis, some of whom have been followed ten years, have signs of Perthes' disease developed in the affected joint. In only 2 of my patients has this hip synovitis syndrome recurred.

The hip joint consists of opposing cartilage-covered bone ends, joined by a flexible tube of dense connective tissue, the articular capsule (table 4). The hyaline cartilage covering the bone articulations is elastic and avascular and has relatively little capacity for repair following injury. It is readily damaged by many toxic agents, by acute injuries, and by minor and repeated trauma. The epiphysis of the head of the femur, the growth apparatus, is spongy bone with a thin cortex of compact bone. The articular capsule consists of dense, fibrous tissue reinforced by tendons, muscles, and fascial layers. The glenoid lip is fibrocartilage. The acetabulum consists of an articular lunate portion and a non-articular portion, the acetabular fossa. The ligamentum teres is an interarticular flat band stretching from the acetabular fossa to the head of the femur.

The normal joint contains a small quantity of cell-poor, clear, pale yellow, viscous liquid.^{6,7} This fluid is probably secreted largely by the synovial cells. It is a dialysate of blood plasma, containing albumin, globulin, sugar, and mucin.

The synovial layer of the joint capsule consists of a single layer of cells, which gives a cobblestone appearance in cross section. Supporting this membrane is a thin layer of fibroblastic cells. The membrane does not cover weightbearing surfaces of the articular cartilage. The synovial cells respond by proliferation when injured by trauma, infection, impairment of blood supply, or alteration of metabolic activity.⁸ One or more of these hip structures is involved in the syndrome of hip synovitis. Though no pathologic material seems to be available, the synovial layer

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TABLE 1
HIP SYNOVITIS IN CHILDREN
DIFFERENTIAL DIAGNOSIS

1. Polio
Guillain-Barré polynueritis
2. Rheumatic fever
3. Perthes' disease
Slipped femoral epiphysitis
4. Trauma
5. Malignancy
6. Sickle-cell disease

TABLE 3
HIP SYNOVITIS IN CHILDREN
REVIEW OF LITERATURE

	Cases	Male : Female ratio	Average age
Miller ¹ (1931)	77	2:1	7.5 yr.
Butler ² (1933)	34	3:1	8.5 yr.
Finder ³ (1936)	22	1:1	5.4 yr.
Edwards ⁴ (1952)	13	1:1	5.4 yr.
Donaldson ⁵ (1955)	52	4:1	5.9 yr.
Tudor (1959)	118	1.3:1	3.6 yr.
Total	316		6.0 yr.

TABLE 2
ORTHOPEDIC CASES

1. Perthes' disease	5
2. Osteomyelitis	2
3. Baker's cyst	6
4. Osteochondritis	4
5. Tuberculosis	2
6. Spondylolisthesis	1
7. External chondromatosis	1
8. Synovitis of hip	118
Total	139

TABLE 4
ANATOMY OF THE HIP JOINT

1. Hyaline cartilage covering bone articulations
2. Epiphysis of head of femur
3. Articular capsule
4. Glenoid lip
5. Acetabulum
6. Ligamentum teres
7. Synovial fluid
8. Synovial layer of joint capsule

of the joint capsule appears to be the involved tissue.

Hip synovitis in children has been variously thought to be due to trauma,⁹ allergy, a stage of Perthes' disease, an organ antibody reaction, and infection. It is my impression that the joint capsule in this entity is involved with a mild antibody reaction to some bacterium.

If untreated, the child with hip synovitis may complain of a painful hip and may limp for three or four weeks or longer. It is possible that, with continued weightbearing, chronic inflammatory changes may occur in the synovium. It has been my practice to treat these children with an antibiotic, such as penicillin or tetracycline, and to insist on strict bed rest with no weightbearing for at least seven days. When there is acute tenderness and pain which does not respond to these simple measures, extension of the leg with traction may be necessary for a few days. In my experience, I have seen no children refractory to this treatment.

SUMMARY

The problem of hip pain in children has been reviewed. The hip synovitis syndrome usually occurs in boys 3 to 8 years of age and is a benign disease which responds to rest and antibiotics.

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Basic Bronchopulmonary Physiology in the Management of Postpolio Respiratory Problems

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PATIENTS WHO SURVIVE the acute attack of poliomyelitis may completely recover or may be severely disabled, often permanently. It is axiomatic that the anterior horn cells that first show dysfunction at the onset of the disease are the ones most likely to be the seat of chronic residual paralysis. Only that paralysis which affects the respiratory system will be considered here. The clinical manifestations in this group include the following:

1. Respiratory muscle paralysis and poor pulmonary ventilation
2. Laryngeal obstruction
3. Difficulty in swallowing
4. Oropharyngeal secretions
5. Tracheobronchial secretions

SUMMARY OF THE CLINICAL PICTURE

Spinal polio, without bulbar involvement. From the standpoint of the respiratory tract, patients in this group are the least difficult to care for. Even if these people require a mechanical aid to breathing, such as intermittent positive pressure, a cuirass type of respirator, a rocking bed, or a tank respirator, their problems are usually not too difficult to manage, except that their ventilation must be maintained. They can eat normally, can handle oral secretions, and, therefore, constitute the least complicated group. They tend to have few respiratory problems, unless there is a tendency for bronchitis or pneumonitis to develop.

Bulbar polio, without spinal involvement. The characteristic site of involvement in bulbar polio is the nucleus ambiguus, which supplies all the muscles of the larynx and pharynx. In addition, any of the cranial nerve nuclei may be involved. In the chronic phase, these problems generally resolve themselves around difficulties in swal-

lowing. Unless the patient can swallow 100 per cent *effectively*, he is potentially in danger at any time. It is distressing to care for patients in whom the ability to swallow does not develop at all, but it is sometimes more dangerous when they improve sufficiently to swallow some food but cannot coordinate well enough to keep a certain amount of food and liquid off the larynx and out of the trachea.

Invariably, a psychosomatic basis is suggested for delayed recovery in such patients. Often heard are such things as "He can swallow his saliva in his sleep, why can't he swallow it in the daytime?" It appears that the ability to swallow is much more a matter of coordination than it is a simple muscular action. We initiate the act of swallowing voluntarily, but it proceeds in a normal peristaltic manner more or less involuntarily. The difficulty that these people have is mostly in initiating the act of swallowing. Some operations have been devised in order to relieve this. However, the over-all results seem to be disappointing. Also, we have dilated the hypopharyngo-esophageal junction in a few patients, not because they have an organic stricture but because, in some way, this seems to help them regain sufficient coordination to swallow. The results of dilations in this instance are equivocal, although it has seemed to help in some cases.

Many of these patients can swallow food in large boluses, for example, bread, meat, and so forth, but cannot handle saliva or other liquids well. In these persons, it is extremely *unsafe* to allow normal oral intake, not because they cannot swallow at all but because they cannot safely prevent aspiration into the larynx and trachea.

Bulbospinal. This type of polio, of course, presents the most complications. If to the problem of faulty ventilation is added the multiple problems of faulty deglutition, it is difficult to the point of impossible to keep oropharyngeal

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secretions out of the trachea, and when they enter the trachea, these patients do not have the ability to cough them away. In most instances, the chronic bulbospinal problem requires not only mechanical aid to respiration but also that a tracheostomy be maintained until improvement is sufficient to prevent saliva from entering the trachea. When one must wear a tracheostomy tube for a long time, a plastic tube is kinder to the mucosa than a metal one.

MANAGEMENT OF SPECIFIC PROBLEMS

Maintenance of pulmonary ventilation. It is axiomatic and even trite, but sometimes we forget that either the patient's lungs must be ventilated or he dies. It is quite true that there is such a thing as acclimatization to decreased pulmonary ventilation. A state of hypoventilation that would take the life of one acutely ill with polio may be quite well tolerated in one who has lived with the disease for many months. As a matter of fact, there are perhaps some disadvantages to hyperventilation by artificial means if continued for long periods, not the least of which may be difficulty in weaning the patient away from the respirator. However, with cautious persistence, most of the chronic patients can be weaned away from mechanical aids.

These people are placed in grave jeopardy by the slightest respiratory infection, because inflammation or secretions in the tracheobronchial tree or lungs interfere with ventilation.

Laryngeal obstruction. It is uncommon for a patient who has recovered from bulbar polio to have a degree of laryngeal obstruction that significantly interferes with his ventilatory capacity, although the voice is often changed. When ventilation is impaired, a permanent tracheostomy or a surgical operation to obtain vocal cord abduction may be required.

Oropharyngeal and tracheobronchial secretions. Tracheobronchial secretions and aspirated saliva represent the gravest problem in the chronic polio patient. If one cannot swallow effectively, a certain amount of saliva invariably enters the tracheobronchial tree. The presence of pre-existing lung disease, such as asthma, chronic bronchitis, bronchiolitis, bronchiectasis, pulmonary emphysema, or other sources of bronchorrhea and hypoventilation, further impair the ventilatory capacity of patients crippled by polio. Any secretions which appear in the tracheobronchial tree must be removed by one means or another. A patient who needs a respirator cannot cough effectively; therefore suctioning is essential, and, practically speaking, in most instances, a tracheostomy is required.

Alimentation and deglutition. Patients who cannot swallow must be fed by other means. In general, plastic nasogastric tubes are preferable to gastrostomy. The incidence of ulceration of the esophagus is very low with a small indwelling plastic tube as opposed to rubber tubes, which seem to cause more irritation. There is, however, an almost 100 per cent incidence of otitis media in patients with a chronic indwelling nasogastric tube. This does not necessarily constitute a major problem, since antibiotics control it fairly well.

It should be mentioned again that there are postpolio patients who can eat solid food but who cannot swallow their saliva, and, in our opinion, oral intake except by tube feeding is fraught with the grave danger of aspiration in this group.

SPECIAL CONSIDERATIONS IN PRODUCTION AND CONTROL OF TRACHEOBRONCHIAL SECRETIONS

Physiologic considerations with reference to atmospheric temperature and humidity. Much has been written about the effects of atmospheric temperature, humidity, and barometric pressure on the tracheobronchial tree. The opinions expressed in this paper are our own and have been developed in the course of management of polio patients and others with respiratory problems. However, there is much precedent for our line of reasoning. We shall make no attempt to review the literature here but have included in the bibliography of this paper several sources of material in which the authors considered these factors. The following is a general outline of this viewpoint:

1. Dampness, or increased atmospheric humidity predisposes to bronchorrhea, bronchitis, and pneumonitis.

2. Cool temperatures plus dampness cause the changes to come about more quickly and render the changes more severe and more dangerous to the patient.

3. A patient who tends to perspire has more difficulty.

4. A patient's interpretation of atmospheric humidity or temperature may be completely erroneous as far as what is physiologically optimum for him. Simply stated, this means that surroundings which the patient considers comfortable may be highly detrimental to him by causing bronchial edema and bronchorrhea.

Specifically, the physiologic effects of cooling, chilling, and dampness are the following:

- a. Bronchorrhea.
- b. Bronchiolar narrowing due to (1) edema

of bronchial and bronchiolar mucosa and (2) spasm of the bronchiolar musculature. In polio bronchorrhea, mucosal edema and bronchiolar spasm mean suffocation!

- c. Cough, or the sensation of desiring to cough, since many of these patients cannot cough.
- d. The combination of bronchorrhea, bronchiolar narrowing, and cough results in a sensation of warmth on the part of the patient, which usually causes him to seek more breeze and less clothing, thus intensifying the derangement, hypoventilation.

Life and death importance to these patients.

In a chronic polio patient who has a borderline ventilatory capacity, anything which alters his ability to ventilate can have a profound effect. Small amounts of mucus, a very moderate degree of bronchiolar edema or spasm, or even just a little bit of debris and detritus can cause a significant respiratory obstruction. There is also a diffusion defect involving segments or lobes with bronchi that are blocked. Even though pulmonary artery flow through an atelectatic segment or lobe becomes progressively less as the situation becomes chronic, this can result in considerable arterial oxygen desaturation.

Year in and year out, chronic polio patients who have been doing fairly well come back into the hospital sick with so-called pneumonia. They are treated for infection alone, with antibiotics as the primary approach, and die. The incidence of these readmissions is significantly associated with cold snaps, cold fronts, "dust storms," and other seasonal changes in the weather which result in falling temperatures. The combination of a drop in atmospheric temperature plus a rise in atmospheric humidity is particularly detrimental to these people initially.

The mechanism is simple enough in analysis, but it is a little bit difficult at times to persuade families of patients and hospital personnel to apply this reasoning to the care of the patient.

Methods of control. Ideally, the patient with a chronic breathing problem, especially one who has contracted a respiratory infection, should be placed in a room of constant temperature and humidity, in a "glass house" so to speak. In general, this is not possible. Even in air-conditioned places, the controls are often inadequate and there are wide variations in temperature in a given twenty-four-hour period. Also, some air conditioners dehumidify well and others dehumidify very poorly. Some are "evaporative coolers" or water coolers which add to the humidity.

Because it is difficult to control atmospheric

temperature and humidity under some circumstances and impossible under others, one must look to other means to keep these patients alive. The most available method is to clothe them in lightweight cotton garments. The maximum protection that can be afforded these patients can be attained with *multiple layers* of simple, inexpensive cotton clothing if properly applied. The basic attire should include lightweight cotton socks, lightweight cotton jersey strap shouldered undershirt, and long sleeved and long legged cotton pajamas. The best results are obtained when *all skin* except hands and head and neck is covered with *garments*.

To apply bedcovers over a naked skin does not provide one with as much protection as the application of a much lighter cotton garment worn as clothing next to the skin.

By all means, antibiotics should be used judiciously in the presence of an infection, preferably selected on the basis of sensitivity studies performed on cultures of the offending organisms. It should be stressed, however, that treatment with antibiotics alone does not result in a quick enough reversal of the vicious cycle of physiologic changes to keep these people alive if they are really in difficulty.

SECRETIONS

Here, as in any other phase of the treatment of polio, secretions that appear in the tracheobronchial tree, whether they be aspirated saliva, the exudate incident upon an infectious bronchitis, or the bronchorrhea caused by a bare skin, must be removed by one means or another. Postural drainage is usually not feasible in a patient who requires a mechanical aid in order to breathe. Certainly, these people should be turned frequently in order to prevent pooling. It is very difficult to aspirate the trachea in polio patients through the normal nasal route that is used for routine postoperative suction. Bronchoscopy is inadequate when done perorally because any method used to remove secretions from these people must be repeated at intervals of minutes to several hours, and it is not feasible to bronchoscope a patient eight to twelve or more times per day by the peroral route. For that reason, a patient who is in severe difficulty must either keep his tracheostomy or have one made in order to control his secretions.

HUMIDIFICATION

A high atmospheric humidity is detrimental to these patients because the usual physiologic effects of chilling become more profound under this condition. On the contrary, a high humidity

—water in a vapor state—is absolutely essential in the inspired air. Also, much better results are obtained when the inspired air is both humidified and *warmed*. Ordinary cold nebulizers, such as the NCG, the Mist O₂ Gen, and many others, with or without wetting agents, may be quite satisfactory in a simple case. However, when the patient gets beyond control, sometimes a near miracle can be wrought by switching to hot steam or an effective nebulizer warmer.

CONCLUSIONS

1. Postpolio patients may have difficulty with the mechanics of ventilation due to respiratory paralysis and the inability to cough; they may have difficulty in handling oropharyngeal secretions due to paralysis of deglutition, or they may aspirate; they may have a narrow airway at the glottis due to vocal cord paralysis, or, they may have bronchorrhea, mucosal edema, or bronchiolar spasm.

2. Any degree of respiratory obstruction endangers the lives of these patients.

3. It is important to take cognizance not only of the infectious aspects of the so-called respiratory infections but also of the physical factors of cooling, chilling, perspiration, wet skin, atmospheric temperature, and humidity and their *physiologic effect* on the respiratory system.

There is an inescapable correlation between changes in the weather and morbidity and mortality rates in patients with a chronic disability incident to respiratory polio.

4. The program of control involves education of the patient, the parents and relatives, nurses, attendants, physical therapists, physicians, and everyone who cares for these people. Nakedness plus a damp skin plus a cool or cold atmosphere endangers their lives. Any carelessness in keeping these people protected from changes in temperature and humidity will reflect itself in staggering morbidity and mortality rates.

5. When possible, constant temperature and humidity should be maintained. In addition, a

striking degree of protection can be afforded by the application of lightweight cotton garments.

SUMMARY

1. An outline of the respiratory problems in chronic poliomyelitis has been presented.
2. A brief discussion of the physiologic derangements in such patients has been presented.
3. Management of specific problems has been discussed.
4. A consideration of the physiologic effects of cooling, chilling, and dampness has been presented, and methods for protection of the patient have been outlined.

We are indebted to Dr. Ivan Danhof of the Department of Physiology and to the librarians, Mrs. Violet Baird and Mrs. Elinor Reinmiller, of the University of Texas Southwestern Medical School for their help in locating the bibliography.

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ACUTE RESPIRATORY disease of unknown origin may affect workers handling compost used in growing mushrooms. Common symptoms are fever, cough, chest pain, dyspnea, nausea and vomiting, and headache. Roentgenographic appearance of the lungs suggests widespread, diffuse infiltration, and rales and tachycardia are noted. Treatment is supportive. The patient must be removed from exposure to compost, since further contact may cause recurrence of the disease.

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A Practical Approach to the Diagnosis, Management, and Treatment of Hypertension

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OVER THE PAST FIFTY YEARS or more, a vast amount of writing has accumulated on the subject of so-called essential hypertension. It is altogether fitting and proper that this should be so, for this disease is one of the most common disorders that plague mankind and can be a vicious disabler, crippler, or killer. Ofttimes, because it is so common and well-known a disorder, those afflicted with it may tend to minimize its importance and ignore it. Such neglect can be a grievous error in that they may thus fail to seek the necessary treatment at a time when damage to their vascular systems can, and must be, prevented. By not seeking treatment, they reach by default, as it were, the stage of advanced, profoundly serious and most often irreversible hypertensive, and often coexistent arteriosclerotic, cardiovascular renal disease. On the other hand, all of us are familiar with the patient who becomes severely neurotic over his blood pressure readings to the point where these readings permeate and affect his every thought and action. Such a patient may become an abjectly miserable person in his worry over a relatively borderline and benign condition which, even if completely ignored, might never progress or do any injury to his vascular system.

And yet, either extreme in attitude may be the proper one, for the patient's physician usually has no way of knowing early in the course of this disease which clinical course his patient's disease will follow! As a matter of fact, the physician cannot even answer the questions that

any patient has every right to ask his doctor, namely, "What causes this ailment from which I suffer? Why did it afflict me? What may I expect it to do to my body?" and, most frequently asked, "What can I do to rid myself of it and prevent its leading to my disability or death?" In each instance, as these questions are asked, and they inevitably are, the physicians must in light of today's knowledge of this disease, or lack of it, answer "I really don't know for certain." In view of this, some sort of practical approach to the handling of this disease in our present state of limited knowledge of its pathogenesis and therapeutic attack may be in order, and, of necessity, such an approach must be wide, elastic, and ready to be changed.

It seems almost unbelievable, and it is certainly frightening that in this age and in spite of the vast amounts of time, effort, and money spent in the search of the cause of this disease, the actual etiology remains unknown. It seems likely, according to Wakerlin,¹ that "essential hypertension is a generic classification consisting of several distinct types of hypertension" rather than its being "a single clinical entity with various degrees of functional alterations in different body systems." It appears that there are "neurogenic, electrolyte, endocrine, and renal changes in pathophysiology that may have pathogenic significance for essential hypertension." The theory that increased smooth muscle tone in the arterioles is the cause of the increased resistance, which, in turn, necessitates a rise in systemic arterial blood pressure in order that blood flow be maintained at the needed level seems to be quite universally accepted. It is the exact cause

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of this increased tone which eludes our investigators. Neurogenic factors, through whatever channels their effects are mediated, are of utmost importance. It seems most likely that a fundamental disturbance of sodium and water regulation is related to the syndrome of hypertension, but, as yet, it is not clear how much of this is cause and how much effect. Certainly, the response of the blood pressure to sodium restriction in the diet or to sodium excretion is anything but constant. That the kidney harbors the answer to at least a part of the riddle could hardly be denied, but outstanding investigators favor different ideas in regard to the exact relationship, with evidences pointing to neurogenic, humoral, and mechanical mechanisms. Advancing age is not a factor in the development of essential hypertension, since its course usually starts in early adulthood and in middle age. It is, of course, true that it rarely begins in early childhood or in the teens and that hypertension at these ages is usually not of the idiopathic variety and that, when it is present in youngsters, it merits an exhaustive search for some disease of which hypertension is but one of the secondary manifestations.

CAUSES OF HYPERTENSION

Needless to say, even in the age groups in which hypertension is 9 or 10 times as apt to be of the essential variety than of the secondary or symptomatic variety, a careful search for other possible types is in order. As pointed out by Chassin,² hypertension secondary to renal disease may have a multiplicity of causes and involves ruling out such possibilities as glomerulonephritis, pyelonephritis, gouty kidney, toxic renal changes, diabetic renal disease, collagen disease affecting the kidney, polycystic renal disease, nephrocalcinosis, amyloid disease of the kidney, obstructive disease of the kidney, and the multiple causes of unilateral renal disease. The search for pheochromocytomas in cases of hypertension has become more commonplace, and Kvale³ and associates have written extensively on the subject, so that today Regitine tests, benzdioxane tests, and histamine tests and chemical determinations of urinary catechol amines nicely uncover these cases. These tests have become increasingly used since it became quite obvious that an adrenal tumor could be the basis for a sustained hypertension and that not only in paroxysmal hypertension had it to be considered. It has been stated that "*every* patient with hypertension should be evaluated, at least once, for pheochromocytoma."⁴

In the younger age groups particularly, aortic

coarctation and intracranial disease must be ruled out as possible causes of hypertension. In the older age groups, only the very benign type of systolic hypertension associated with the decreased elasticity of the aorta should be excluded from any practical consideration of hypertensive cases, since it is of little importance and merits no treatment. Cases of thyrotoxicosis, arteriovenous fistulae, and so forth, must also be eliminated, as, in such instances, the treatment is directed toward the underlying disease. It is likely that more cases of aldosteronism⁵ will come to light in the years to come. At this time, the possible relationship of this disease to hypertension is not known, but some have suspected that a state of mild hyperaldosteronism might be etiologically related to human hypertension.⁶

What then are some of the practical diagnostic approaches? Obviously, one of the first considerations is the establishment of a level for both systolic and diastolic readings, which, if exceeded, would justify a diagnosis of hypertension. Needless to say, the arbitrary level for upper limits or normal of 140/90 or 150/100 is as unrealistic as the setting of the level for a normal reading at 120/80. Chassin² states that the finding of a "persistently" elevated diastolic pressure of over 90 mm. of mercury means hypertension. Master and co-workers⁷ on the other hand insist that "the accepted limits are too low and must be liberalized." These authors feel that while, at age 16, the lower limit of diastolic hypertension should be considered as 90 mm. of mercury, the same limit at age 64 should be set at 110 mm. of mercury. They also pointed out that, at ages 55 to 59, the lower limit of systolic hypertension is 180 mm. of mercury in the male and 185 in the female. In the younger age groups, the limits of normal are lower in the female. At about age 40, the limits in both sexes are about the same, and thereafter the upper limits of normal are somewhat higher in the female. They quoted studies by Alvarez as well as by Diehl and Sutherland to point out that, in large numbers of college students studied, from 11.5 to as high as 22 per cent had systolic blood pressure readings in excess of 140 mm. of mercury. They felt that it would be a very good idea to return to the use of the old "rule of thumb" whereby the normal systolic blood pressure averages about 100 plus the person's age. It would seem that, in general, the setting of the following limits would be justified: at age 25 or under, the blood pressure should not exceed 145/95; at ages 25 to 45, the blood pressure should not exceed 160/100; and past the age of

45, the blood pressure should not exceed 170/110. Obviously, readings at or just below these levels fall into the borderline group.

With these thoughts in mind, the incidence of hypertension in the general population is in the neighborhood of 6 per cent,⁸ a high enough level to make this one of our most common diseases, but still below the often quoted figures of 20 to 25 per cent. Perera points out that hypertension usually "starts earlier and lasts longer" than is generally suspected, averaging about twenty years in duration, and is ordinarily detectable in early adult life, is more common in the female, and seldom starts after the age of 50.

PROGNOSIS

From a practical point of view, it is also important to be able to attempt to prognosticate for the hypertensive patient. The increased incidence in women has long been known, and the tendency for the disease to have a more benign course in women is substantiated by all observers. It is also apparent from all statistical studies that the disease is more severe in patients in whom it develops at a younger age. Frant and Groen⁹ have stated that the prognosis is worse in men than in women and that the younger hypertensive patients do not do as well as those in whom the disease develops later in life and that coexistent heart disease, albuminuria, or diabetes reduces the life expectancy. They stated further that the life expectancy was directly related to the levels of both systolic and diastolic pressures. They, as well as others, also felt that the ophthalmoscopic findings, as described by Wagener and Kieth,¹⁰ have "great prognostic reliability." As a matter of fact, most writers on the subject of hypertension feel that fundus findings allow a greater accuracy in evaluating the status and prognosis of this disease than any other single symptom or sign, including the level of the blood pressure itself. It appears that the hypertensive patient who is obese has a better prognosis than those who are lean or of normal weight. This might be due to the fact that the obesity per se, rather than underlying hypertensive disease, is at least in part responsible for the elevation, while, in the nonobese person, the elevation is due solely to the disease process itself.

A recent study was done on the prognosis of 290 patients who had essential hypertension prior to 1944. The survivors were restudied in 1950 and in 1957, and, in those who died, the causes of death were determined. It was found that the causes of death were, in order, cerebrovascular complications in 55 per cent, cardiac complications in 24 per cent, renal complications in

12 per cent, and miscellaneous conditions in 9 per cent. It was of interest that, in this series, some sixteen years after discovery of the hypertension, slightly over half of the patients were still alive and had had no "special antihypertensive treatment." Better prognoses were found in those with labile hypertension, in women as compared to men, in those whose blood pressure decreased with sedation or rest, in those with normal electrocardiograms and chest x-rays, in those with less fundus changes, and in those with systolic pressures under 200 mm.¹¹

Certain associated findings and phenomena are of great practical value in properly assessing the state of the disease. As mentioned, one of the most significant aids in evaluation is the ophthalmoscopic examination, and it is most important for the physician to acquaint himself with the art of this examination and apply it to all patients but to those with hypertension in particular. The electrocardiogram is, when positive, of value in assessing the amount of strain on, or hypertrophy of, the left ventricle of the heart in hypertension. It is not a specific test, of course, since other disorders placing a strain predominantly on the left ventricle, such as coarctation of the aorta, aortic stenosis, and so forth, give a picture that is generally indistinguishable from the strain pattern of hypertension. Occasionally, an established left ventricular strain pattern improves or even disappears with reduction of blood pressure to more normal levels. However, Helmcke and associates¹² pointed out that the appearance of the electrocardiogram before institution of therapy had no *predictive* value on the success or failure of the therapy. They pointed out also that "there was no consistent relationship between height of the blood pressure, total severity indices, size of the heart by teleoroentgenogram, and the electrocardiographic changes attributable to left ventricular hypertrophy. Thus, while electrocardiographic estimates follow group trends rather closely, they do not accurately reflect the cardiac or even the over-all status of individual patients with hypertensive heart disease."

The chest x-ray affords by far the most accurate measure of the exact size, shape, and position of the heart. The seriousness of the hypertension is, in part, judged also by how it has affected the heart size, since this cardiac hypertrophy is the forerunner of cardiac dilation and then further hypertrophy and dilation until, ultimately, congestive heart failure may supervene. On occasion, the successful reduction of blood pressure levels is accompanied by a reduction in heart size on the x-ray, though, in advanced

instances of cardiac enlargement, this is seldom of a noteworthy magnitude. The level of the blood urea nitrogen is of inestimable value in judging the severity of hypertensive disease. When it is elevated, it serves as a prognostic sign of value and also helps in deciding what type of therapy is indicated or, more important, contra-indicated, as will be discussed later. Another diagnostic and prognostic aid, though not often available and not recommended as a routine measure, is renal biopsy, which can be obtained at the time of sympathectomy. There is a "general correspondence between the degree of renal arteriolar sclerosis and the clinical evaluation, postoperative blood pressure response, and renal function judged by PSP excretion tests. Severe arteriolar sclerosis was associated with a higher mortality."¹³

It is noteworthy that at this time, in contrast to what was the case some ten years ago, a discussion of prognosis even in malignant hypertension is justified.¹⁴ Until these people were treated with presently available potent drugs, the prognosis was that 80 to 90 per cent of such patients would be dead within a year. In this study, however, 70 per cent of patients survived the first year, and 32 of 84 patients, or 38 per cent, were alive after seven years. The authors emphasized that their study and others showed that "the syndrome of malignant hypertension is reversible by any procedure that results in prolonged lowering of diastolic pressures toward normal levels." In this type of hypertension, renal failure is the most common cause of death, the remaining deaths being due to complications of arteriosclerosis.

It would appear that the diagnosis of essential hypertension is one of the simplest to make, since it is dependent not on the diagnostic skill of the physician but only on a simple measuring device, the technic of which can be taught quickly to students, nurses, ward aids, and even to the patient himself. This, however, is not the entire case. It is true that if one follows the rules set forth in any book regarding the method of measuring blood pressure, he is very apt to obtain the correct reading. In many cases, however, the readings can be quite wrong, often by a considerable number of millimeters, if the determination is not made carefully.

TECHNICS IN DETERMINING BLOOD PRESSURE

The cuff should be smoothly applied in the correct position on the arm. The cuff should be inflated tightly enough to completely occlude the brachial artery and then be deflated at the proper rate of speed—not so slowly that the pa-

tient's arm hurts from the overly long compression nor so rapidly that the reading for the first sound coming through can be "off" by 10 to 15 mm. or more. Several readings should be made with a few minutes rest between readings until a fairly stable level is reached. Both the highest and the lowest readings should be recorded, so that a measure of the lability of the patient's pressure is available. One should become familiar with the relatively simple method of reading the systolic pressure and the somewhat more difficult recognition of the end point denoting the diastolic pressure. One should be alertly aware of the variability of the impulses coming through under the cuff in cases of atrial fibrillation, multiple premature systoles, and others of the arrhythmias, which make the accurate readings more difficult, and of the need for taking these readings more slowly and infinitely more carefully.

The examiner should know whether his patient rushed to keep his appointment and had been harassed before coming or if he arrived in a leisurely manner and spent a pleasant and brief wait in the doctor's reception room. Obviously, the examining physician can not be aware of all the possible irritating, frustrating, or frightening occurrences that could have immediately preceded the patient's visit and have affected his blood pressure, but he can, if he bothers to observe the patient and his manner, usually tell whether or not he is at ease. Engaging the patient in a conversation about a nonmedical topic while the cuff is being applied and inflated can do so much to divert his attention and relax him, thus setting the stage for a more accurate reading. It is occasionally possible, if the examiner has good ears and is a keen observer, to take the pressure quite accurately while the patient is speaking, thus almost completely sidetracking his attention from the procedure at hand.

The physician should also recognize the patient who perspires, trembles, and runs a tachycardia of 125 per minute or over at the mere sight of the apparatus used to take blood pressure and in whom a blood pressure even remotely akin to his "resting" level is impossible to take. Schroeder and Perry¹⁵ pointed out that "when the cuff is wrapped about the arm, intense neurogenic vasospasm may occur before the examiner has had time to inflate it." They quoted Janeway to the effect that "the act of compression of the cuff may raise blood pressure." In such people, periodic observations of the blood pressure may do more harm than good, and profound sedation may be essential for any sort of accurate evaluation.

An oppressively warm examining room can make a patient feel faint and result in a false low reading, while a cold room can affect the blood pressure reading so that it will be incorrectly high. The patient's body temperature and the state of his peripheral circulation may, of course, have a distinct effect on his blood pressure. The doctor would do well to know whether the patient has any discomfort or pain while he is being examined and whether he has recently taken any drugs which could have either raised or lowered his blood pressure. The physician should also be sure that the patient's arm is in a comfortable position and at rest, so that the muscular tension of the arm does not produce falsely high readings. Other observations, such as taking the blood pressure in both arms and in the legs, in both lying and sitting positions, before and after sedation, before and during immersion of the hand in ice water, and in other special situations, should be considered in all patients. At least the possibility of taking these special readings should be kept in mind, so that they will be taken when indicated.

Weather variables, such as barometric pressure, air temperature, and relative humidity appear to have no effect on the blood pressure¹⁶ and need not be considered as possible factors in evaluating blood pressure levels.

Also of great practical importance is the information that is given to the patient about his blood pressure. There are physicians who feel, and probably correctly so in many respects, that there is no one to whom the knowledge of his condition is so important as to the patient and no one who has a greater right to know his exact condition than the patient. On the other hand, there are equally well-informed physicians who insist that the exact blood pressure readings to most patients can be nothing more than confusing and frightening numbers with no real meaning. These men insist that they will inform the patients of their general condition and treat them as indicated without giving them a list of numbers to mull over.

On the other hand, to the patient, high blood pressure means too high a "number," and he can't understand why he can't follow his own progress by knowing what his "blood pressure should be" and what it is on each visit to his doctor's office. He asks why he can't judge the adequacy or inadequacy of his response to therapy simply by the way in which his blood pressure compares to the "normal" each time it is taken. He is, for the most part, unaware of all the emotional factors involved, of the unimportance of a single reading, of the marked varia-

bility that readings may show without indicating changes in his fundamental condition, of the need not to judge a medication by lack of an immediate response, and, most important, he could not possibly be aware of either the normal vascular physiology or of the pathology and pathologic physiology of hypertensive disease. The doctor should not, however, hide his own lack of knowledge about hypertension and its responses by saying to his patient in a somewhat supercilious tone, "This is all too involved for me to explain it to you. You must trust me and not ask me what your blood pressure is each time you come." He would establish a much better rapport with his patient if he set aside a half hour to explain to him what is as well as what is not known about hypertension. He should make it clear to the patient that the over-all clinical picture is the important thing and that the blood pressure reading is only one facet of the over-all picture, others being cardiac status, blood urea nitrogen level, electrocardiographic evidences of heart strain, roentgenographic evidences of cardiac enlargement, the presence of abnormal elements in the urine, the presence of vascular changes in the eyegrounds, and the presence of early signs of left ventricular failure.

It should be pointed out to the patient that the way he feels in general is an important index of how he is getting along, although all physicians are well aware of the fact that the patient usually cannot tell by the way he feels when his blood pressure is normal and when it is high. Of course, headaches, dizziness, visual disturbances, and so forth, of hypertensive encephalopathy are symptoms of severe blood pressure elevation, but aside from these situations, the patient often feels no different with a blood pressure of 150/90 than he does with one of 230/125, assuming that all other variables are unchanged. How often does the physician see a patient who is certain his blood pressure is "better today because I feel so good," only to find that the pressure that day is unusually high? It is equally true that a patient may say that his blood pressure must be "terribly high because I feel so bad" and then have one of his better pressure readings. In general, it is probably true that a hypertensive patient under active antihypertension drug therapy who does not feel well is much more apt to be so indisposed because his blood pressure dropped too low or as a result of side effects from the drugs than from his pressure being too high. The careful explanation of this to the patient will do much to increase his confidence in his physician and lessen his in-

sistence that he can judge his progress if only he is provided with the pressure readings.

WHAT TO TELL THE PATIENT

The doctor must reply in some way to his patient's question regarding his blood pressure readings. Just what should the doctor tell the patient in order to satisfy him, to reassure him, and, above all, not to frighten him? How bound is the doctor to tell the patient the truth about each reading? Patients are accustomed to knowing, for example, the level of their hemoglobin concentration, and physicians do not hesitate to acquaint the patients with their progress in treatment of anemias by telling them how high or low the blood count is. The diabetic patient gauges the adequacy of his control by his urine sugar tests, which he ascertains himself, and by his occasional fasting blood sugar levels, which his doctor usually gives him without hesitation. The hypertensive patient, too, in spite of almost any explanations given him, will ultimately be most contented if he is told what his blood pressure readings are. The vast majority of patients assume that if the doctor does not want to give them the readings, it can only be because they are bad.

What then can the patient be told? A good scheme seems to be to give the patient a reading at each visit, which may or may not be correct. If the patient is quite apprehensive and if he is conscientiously following the doctor's instructions, a figure somewhat below the one found is probably best. It is surprising how often the patient relaxes after being told that his pressure is better, and the second blood pressure reading is anywhere from 10 to 30 mm. lower than the first. The second reading, taken without giving the patient this reassurance, is also often lower than the first but usually not to such an extent. If the patient is negligent in taking his medications and following prescribed activities, it is wise to give him the highest readings with a stern warning of possible implications and complications. Obviously, the patient's temperament has much to do with what he should be told and how it is told to him. In the event that the patient is given a reading *other* than the correct one, it is important for the doctor to record on his chart both the blood pressure reading obtained as well as that given to the patient, so that, on subsequent visits, the doctor remembers what he has said. This procedure usually works out very satisfactorily, and, as the pressure decreases, the gap between the actual reading and that given the patient can be narrowed so that finally both are the same figure.

When a patient is hospitalized and the nurse takes blood pressure readings regularly, the problem of what to tell the patient inevitably arises. In these cases, blood pressure variabilities are magnified because of the varied personnel taking the readings, the various extraneous factors related to hospital procedures and their relationship to the time of the pressure reading, and the attitude and manner of the nurse taking the reading. The patient cannot understand all of these factors, and, if he is told each time what his reading is, utter confusion will result. It is easy to avoid telling him what his blood pressure is by explaining to him that hospital rules preclude the nurse giving him this information. He is told that his physician will give him a daily report. It is not wise to tell the patient that the doctor has forbidden the nurses to discuss his blood pressure with him because he will be resentful and may conclude, often erroneously, that his blood pressure must be dreadfully high. The doctor should take the blood pressure himself at each of his visits, first, because the hospital personnel may not take it as carefully, and, second, because, assuming that the pressures taken by the nurses are correct, the doctor's reading will give an idea of what his personality does to his patient's blood pressure. If the doctor's own reading is always much higher than that obtained by the nurse, the patient is either one who invariably reacts in such a way to a doctor's presence or one who reacts in this way to *his* doctor's presence. If the latter is true, the doctor may not have spent enough time or energy to reassure his patient and allay his fears.

A few practical suggestions are in order regarding the patient who goes to a physician other than his own for a blood pressure determination. If he comes in just to check on the report that his own physician has given him in regard to his blood pressure, it would be wise to gently but firmly explain why two readings taken by different men could vary widely and to refuse to take the pressure, urging the patient to return to his own doctor. This is especially true if the patient is under treatment. If the patient wishes to change physicians, this is his prerogative, and, under these circumstances, the new physician must reevaluate the problem and should obtain from the first physician a report of the antihypertensive drugs the patient has been given and what their effects have been. If, as occasionally happens, the patient is a transient or a visitor, he should be told *before* his pressure is taken to expect that, with an unfamiliar examiner, his pressure will probably be considerably higher. He should then be checked and told his pres-

sure, preferably after first ascertaining what his last reading was. His treatment should not be changed unless he is obviously not well or unless serious blood pressure complications seem to be pending.

Aside from the drugs used, the general management of mild and severe hypertension is the same and consists of such measures as rest, reduction of work load, weight reduction, establishment of good general health, and moderation in work, play, and habits. These goals, which, in essence, constitute the goals that any person should strive for, are of increased importance to any hypertensive person.

INDICATIONS FOR DRUG THERAPY

At what point antihypertensive drug therapy should be instituted is one of the most practical decisions that the physician treating hypertension must make. The age of the patient is certainly one factor, and the younger hypertensive patient should be treated more promptly, since his prognosis is not as good as that of the older person with hypertension. The duration of the disease may enter into the decision in some cases. This is especially true in a patient whose blood pressure has been moderately elevated for many years and in whom the disease has not progressed. The use of antihypertensive drugs of any consequence in such a case would seem rather pointless, since time has well attested to the benignity of the disease. On the other hand, another patient with no higher a level but of recent discovery might well *have* to be treated, for it would be unwise to let him go without drug therapy to see if his disease would remain benign. Certainly, on the basis of what is known about the different prognosis in the two sexes, men with hypertension merit earlier and more vigorous drug therapy than do women. One might well treat specifically a man with a certain blood pressure level and not treat a woman with the same blood pressure level. Electrocardiographic, roentgenographic, or urinary changes could well influence the institution of treatment in patients whose blood pressure level does not appear to be too high. Clinical symptoms of renal, cerebral, or cardiac involvement are indications of the urgent need for therapy. It should here be pointed out that, in such cases, too vigorous therapy is to be discouraged in order to avoid, by dropping pressures too fast or to too low a level, increasing renal insufficiency by reducing effective filtration pressure or promoting cerebral or cardiac infarction by thrombosis as a result of the reduced pressure in sclerotic arteries.

The presence of changes in the fundus would indicate that therapy with drugs is in order even if the level of blood pressure does not appear to be very high, since these cases may be examples of latent hypertension.¹⁷ Occasionally, the decision regarding institution of drug therapy may be helped by submitting borderline cases to a cold pressor test,¹⁸ since it has been pointed out that essential hypertension probably affects only subjects who are hyperreactors and that clinically significant hypertension will subsequently develop in the majority of such hyperreactors. A family history liberally studded with strokes, uremia, arteriosclerotic disease, and deaths at an early age should motivate one to start drug therapy with no delay in even the mild case of hypertension. The emotional characteristics of the patient should be considered, and it is usually necessary to institute more active therapy sooner in a tense person, since he is not apt to adapt well to long periods of observation while seemingly "nothing is being done" for him. On the other hand, the relaxed person sometimes merits the beginning of specific drug therapy even earlier than the excitable patient, for his disease is more apt to be serious and sustained than the more labile and variable hypertension of the emotionally unstable individual.

The patient's occupation may also dictate to some extent the need for the institution of therapy. If he has a relatively easy job in which he is happy and relaxed, he may have less need for more active therapy than if he worked at a tension producing job wherein strict time schedules must be met and so forth. The presence of symptoms per se is not an indication for beginning more specific therapy unless it has been established that the symptoms are due to the hypertension. Perera⁸ has pointed out, for example, that about one third of the hypertensive patients in his series *never* had headaches. It is quite obvious to any physician who treats hypertensive patients that the level of the blood pressure is often unrelated to the degree of symptoms and that the degree of symptomatic relief in cases that are treated is generally much greater than the degree of blood pressure drop. Occasionally, marked symptomatic relief may occur without any drop in blood pressure or even in face of a blood pressure rise.¹⁹

The actual level of the blood pressure after it has been established by a number of readings under ideal conditions for observation is then the final determinant of when therapy with specific drugs should be instituted. As stated previously, other factors may be equally or even

more important, but, assuming that they do not exist at all, the pressure itself may be high enough to merit being treated. Each physician usually has his own figure in mind, but it seems fair to say that, in uncomplicated hypertension, specific therapy is indicated in young people whose pressures exceed 150/95, in middle-aged people whose pressures exceed 175/105, and in old people whose pressures exceed 190/110. Levels from about 10 mm. below and up to these levels might be considered borderline.

If, after a painstaking and exhaustive study of the multiple factors involved, one still can not be certain whether a specific therapy is indicated, for instance, in a borderline case, one might be guided by Perera's statement that "the results to be obtained must be balanced by the relative risks of the method employed."²⁰ He stated further that "there is no convincing proof as yet that any therapeutic measure has lengthened the life of a hypertensive individual in the *uncomplicated* phase" and still further that "the level of blood pressure may not be a major factor in the development of the complicating pathologic changes." This suggests that, in borderline cases, drug therapy might well be deferred. Measures of general management and simple "psychotherapy" may often obviate the need for stronger treatment, since, as pointed out by Wolff and Wolf,²¹ an air of "interest by the physician in the feelings, attitudes, and life situations of the patients with essential hypertension reduced or eliminated symptoms in about two thirds" and that, in as high as 20 per cent, the blood pressure was "lowered to normotensive levels for significantly, if not indefinitely, long periods."

When specific drug therapy is started, it should be continued for a long time. The reduction of the blood pressure to satisfactory levels does not indicate a cessation of therapy; this would result in a prompt return of the blood pressure to pretreatment levels if the reduction was due to the medication employed. Adjustment of the medication according to the response and to the possible appearance of side effects is, of course, constantly necessary. A good response is no guarantee that it will be maintained with the same drug or drugs in the same dosages, so regular observations must be made. Therapy should be continued for several years at least, and, if the result has been constant and good, one might then be tempted to reduce the drugs further and possibly discontinue them. One should be prepared to reinstitute therapy at once if the blood pressure again rises. In light of current observations and knowledge, it seems likely that the

drugs presently in use may control blood pressure but do not cure it or arrest permanently the mechanism, whatever it may be, that causes the hypertension. However, some authors believe that it is possible that the basic process of hypertension is actually being reversed in well-treated patients.^{22,23}

From the practical standpoint, the goal to be achieved in the treatment of essential hypertension is simply the reduction of the blood pressure to a normal or near normal level. The method or methods employed to achieve this goal matter little as long as they carry with them little or no risk of making the patient worse or of producing complications or side effects. Obviously, the reason that a reduction in blood pressure is to be desired is that the morbidity, mortality, and complications of this disease are also reduced.

GENERAL TREATMENT

The general measures and some of those that are more specific other than the use of antihypertensive drugs will here be briefly discussed. As mentioned before, the importance of observing the general rules dictated by common sense in the maintenance of general good health must be emphasized to the patient. He should receive adequate rest each night and take a mid-day rest when feasible. He should avoid fatigue and unusual, especially long-sustained, physical exertion. He should reduce his weight if he is obese and, in any event, avoid eating excessively large meals. He should avoid excessive use of alcohol, tobacco, and coffee. Regular exercise should be encouraged; strenuous exercise should be avoided. He should have regular physical examinations to detect possible complications at an early stage and to detect other disorders that might be present and adversely affect the blood pressure. He should regulate his work insofar as is possible to avoid unusual stresses, either physical or emotional. He should make a similar attempt to regulate his social activities. He should be encouraged to develop a hobby, though this should not necessitate great outputs of energy or involve him in competitive activities. His family should be made aware of the role it can play in helping him achieve better general physical health.

In the matter of diet, the major consideration is probably the avoidance or correction of obesity. Low-fat diets need not be stressed unless high blood fat levels are found or arteriosclerosis is manifest. The avoidance of protein foods, advocated so often several decades ago, is fortunately no longer in vogue. The question of salt restriction in the absence of fluid retention or

frank heart failure remains, at this time, incompletely answered. There appears to be no doubt that sodium chloride metabolism is abnormal in essential hypertension and that patients with this disease often have slightly elevated serum sodium concentrations.²⁴ It is not clear exactly what role this disordered salt metabolism plays in the development or perpetuation of this disease, although it has been shown experimentally that salt administration can produce a form of hypertension and salt restriction can reduce the blood pressure in some instances of hypertension. At the present time, it does not seem justifiable to rigidly restrict salt in uncomplicated hypertension and that, with the advent of some of the newer drugs which promote the excretion of sodium, such restriction is even less necessary and, occasionally, may even be harmful.

The treatment of hypertension with extensive sympathectomy should be considered in some cases. Adson and co-workers²⁵ made this concept of treatment popular in the early thirties and credited the idea of resection of the splanchnic nerves in the treatment of hypertension to Daniélopou in 1923. Smithwick²⁶ decries the fact that splanchnicectomy is often "regarded as a measure to be employed as a last resort." He feels that this should be considered as the treatment of first choice in the management of patients "in an accelerated phase of hypertensive vascular disease" as well as in patients of both sexes in that which he classifies as grades 2 and 3 hypertension. He stated further that, even if the surgery is not entirely successful in reducing the blood pressure, it makes the patient more amenable to medical treatment. It is well to remember that the surgical treatment is not without its possible sequelae, and the most common of these are paralysis of ejaculatory powers, sterility in the male, and, occasionally, pronounced fall in blood pressure and tachycardia in the upright position.²⁷ The latter two conditions usually correct themselves with the passage of time. When successful, surgical sympathectomy produces excellent results, but it fails to relieve hypertension permanently in a majority of cases.²²

Sedative drugs certainly have a place in the management of the patient with essential hypertension, and the most simple and effective of these are the barbiturates. These are considerably less expensive and have far fewer toxic side effects than the multitude of so-called tranquilizing drugs, and their prescription for routine use is preferable. The dosage should be adjusted to give mild sedation only, and the use need not be daily or regular but determined by the pa-

tient's day to day needs. A mild bedtime sedative when needed is advisable, and its use need not be discouraged in patients in whom insomnia is a prominent symptom. Chloral hydrate may be substituted in appropriate dosage for either daytime or bedtime sedation, as may the bromides. In prescribing the use of the latter, it must be borne in mind that certain side effects, especially dermatologic, are not uncommon and that, in any event, the continuation of these drugs for over ten to fourteen days without interruption can cause accumulation in the blood stream and a clinical picture of bromidism. Although the action of the Rauwolfia derivatives may be at least in part due to their sedative effect, they are not included here and will be mentioned later.

Bearing in mind the foregoing remarks regarding the nonspecific treatment of hypertension and before entering into a discussion of the present day specific drug therapy, it would be well to point out the difficulties in evaluating the response to any form of therapy used in hypertension. It is noteworthy that in almost every series reported, regardless of the treatment used, the results are favorable. Every author has pointed out that the emotional aspects in the doctor-patient relationship have much to do with the extent of the response and that this factor does not easily lend itself to objective evaluation. It is further noteworthy that in all series reported wherein placebos were used, a favorable response to placebo drugs alone was reported to a greater or a lesser degree in each instance. Ayman¹⁹ used dilute hydrochloric acid drops as a placebo, having chosen this agent because of its bitter taste and because it obviously had no antihypertensive action. It was tremendously revealing when he pointed out that 82 per cent of the patients so treated had marked general improvement, that some complained of recurrent symptoms when the number of drops was reduced, and that side effects even developed in some patients who were unusually prone to suggestion.

Page and Corcoran²⁸ have evolved a method to better evaluate the patient's response, which consists of establishing a relatively lengthy control period in the hospital and then, after specific therapy is instituted, averaging the patient's blood pressures taken twice daily for a week so that a single weekly figure is obtained, helping to iron out the usual "peaks and valleys" and providing a "single datum for a week's effort." In addition to the use of the pressure itself as a guide to the degree of response, they also evaluated the cerebral, renal, and cardiac functions

and their responses to therapy. Obviously, this type of evaluation of response is ideal, but, as the authors pointed out, it is too expensive and otherwise impractical for routine use.

TREATMENT WITH ANTIHYPERTENSIVE DRUGS

And so we come to antihypertensive drug therapy. Extensive reports have appeared repeatedly regarding each of the currently used drugs in respect to their chemistry, pharmacology, mode of action, site of action, effectiveness, toxic effects, and so forth. These will, in general, be disregarded and only a practical approach to dosage, major side effects, and indications for their use will be outlined. Page²⁹ states that a "major breakthrough" has been achieved in lowering elevated blood pressures, but he points out that the "advertising claims for the antihypertensive drugs have, in many instances, been preposterous." It has also been generally deplored that so many antihypertensive drugs have appeared and are continuing to appear in combinations that physicians using these combinations do not have an opportunity to evaluate the responses of the patients to the individual drugs or to adjust the dosages of *each* drug as indicated. The doubtful benefit to the patient of having to take only 1 pill instead of 2 or 3 at one time is far outweighed by the disadvantages inherent in any "shotgun" type of medicinal combination. The list of drugs now in common use is growing rapidly, and the physician should become acquainted not only with the dosage schedule of the drugs but with their side effects and contraindications *before* using them. It is probably quite true that, in general, the more effective the drug, the more apt it is to have side effects and that, as many authors prophesy, most of the drugs in use today will not be in use ten years hence. In the use of antihypertensive drugs, the old adage that one should not be "the first to try the new drugs or the last to discard the old" is very applicable.

Wilkins²⁵ has pointed out that, in using the antihypertensive drugs, one should start with the milder drugs, add to these as needed until the desired blood pressure reduction is obtained, keep the patient on this dosage schedule for from three to six months, and then reduce the dosage to the minimum level to which it can be dropped and still maintain the effect. The general idea of this program is to use the mildest drugs that will be effective and for only as long as is essential. He suggests that the order in which the drugs should be used is as follows: Rauwolfia derivatives, Veratrum compounds, hydralazine, and hexamethonium.

The presence of renal insufficiency, as demonstrated by elevation of the level of blood urea nitrogen, has some bearing on the use of the antihypertensive drugs. Decided lowering of the blood pressure in cases complicated by uremia can only hasten the patient's decline and should be avoided²² unless the uremia is not severe. One should also be cautious in too rapidly or too drastically reducing blood pressure in the presence of cerebral vascular disease, since, although this may well reduce the incidence of cerebral hemorrhage, it may well increase that of cerebral vascular thrombosis. Ganglionic blocking agents are "absolutely contraindicated in recent coronary or cerebral thrombosis and advanced renal insufficiency."²

One of the drugs which enjoyed a moderate degree of popularity some years ago and is still used to some extent is potassium thiocyanate. Kerkhof³⁰ likes to use thiocyanates in cases in which headache is a marked symptom. He points out that blood levels must be checked regularly in order to avoid toxic levels of over 12 mg. per cent. Toxic side effects include the very occasional thiocyanate goiter, dermatitis, and anemia and the more frequent stupor and mental symptoms which occur with higher blood levels.

The Rauwolfia derivatives are considered by most investigators to be the mildest of the currently popular antihypertensive drugs, and some authors believe that their sedative or tranquilizing effect is the major mechanism through which they work. In relatively mild cases, they may be the only drugs necessary, but, as pointed out by Wilkins,³¹ their "main usefulness for long term treatment is as a preparatory, adjunctive agent to be given should there be need for stronger antihypertensive drugs." These are added to the Rauwolfia, not substituted for it. Winsor³² studied the various Rauwolfia alkaloids to determine their comparative effects. He found that reserpine at an average dose of 0.5 mg. per day and the Alseroxylon fraction at an average dose of 4.5 mg. per day were about equal in their hypotensive effect and their incidence of side effects. Deserpidine produced somewhat fewer side effects than reserpine at the same dosage level but produced more nervousness and insomnia when given at night. Rescinnamine in an average dose of 1.5 mg. per day had the least hypotensive effect of the 4 drugs tested.

The toxic effects of Rauwolfia consist of nasal congestion, lethargy, depression, dreams, diarrhea, impotence, and aching muscles. Most of these side effects can be controlled, but the depression is one of the more troublesome and was found to occur in 26 per cent of a recently

reported series. Severe depression developed in about 10 per cent of the patients in this series. It was also pointed out that 50 per cent of persons with a previous history of depression had a recurrence with Rauwolfia medication. Other important points were that no depression occurred in patients taking less than 0.2 mg. of reserpine daily and that, in 11 per cent of their patients, depression developed after a year of treatment. These authors concluded that, whenever possible, the use of Rauwolfia should be avoided.³³

Syrosingopine, a synthetic compound derived from reserpine and recently introduced for clinical use, appears to produce results as good as those obtained with other Rauwolfia derivatives with fewer side effects.³⁴ The dose varied from 1.0 mg. to 4.0 mg. per day in divided doses. It was of great interest that a number of patients who were intolerant of reserpine because of severe nasal congestion, nightmares, severe mental depression, and abdominal cramps were able to take syrosingopine. In patients who were intolerant to this drug also, the side effects were the same as for the other Rauwolfia derivatives.

The Veratrum preparations are probably the next to be used, although, in so many cases, the effective dose is so close to the toxic dose that many investigators do not regard it with much enthusiasm. Nausea and vomiting are probably the outstanding side effects, which become increasingly evident with long continued use.³¹ It may cause bradycardia, as may the Rauwolfia preparations, so that it may counteract the tachycardia that occasionally occurs with hydralazine. In the form of Veriloid, it may be started at a dose of 1.0 mg. three or four times a day and increased to three or four times that dose as tolerated. Protoveratrine A and B in the form of Provell Maleate can be used in a dosage of 0.2 to 0.5 mg. after each meal and gradually increased in dosage until the blood pressure response is adequate or until side effects appear. Some authors feel that this drug is safer and less toxic than the other Veratrum preparations. Few doctors use Veratrum preparations alone. They are most commonly combined with Rauwolfia alone or with Rauwolfia and hydralazine.

Hydralazine is quite freely used, and many writers suggest that it is the next drug to use after the Rauwolfia preparations. Whereas side effects are not uncommon with this drug, they are seldom significant in dosage levels of less than 200 mg. per day.³¹ Since the average dose of this drug in the form of Apresoline is 100 to 200 mg. per day, it is seldom found to be toxic. In cases in which doses anywhere from 300 to

800 mg. per day are used, one must watch closely for toxic side effects. Some investigators say that as time goes on, patients on hydralazine therapy combined with other drugs can often reduce the dosage of hydralazine and maintain their improvement.²² At a daily dose of 400 mg. or over, a lupus erythematosus-like syndrome develops in 10 per cent of patients.² It has been frequently pointed out that hydralazine causes tachycardia and increases cardiac output and stroke volume output.^{2,31,35} Because of this, it has been stated that hydralazine may cause angina, may precipitate heart failure or make it difficult to control, and may cause intolerable palpitation. Either Rauwolfia or Veratrum may partially negate these effects. Page²⁹ feels, however, that too much attention has been paid to these possible side effects, and he points to the work of Crumpton and associates, who showed with this drug an *increase* in coronary blood flow and a *reduction* in coronary vascular resistance without significant change in myocardial oxygen consumption or in cardiac work.

Chlorothiazide, since its introduction in 1957, has enjoyed tremendous popularity. It can cause hypokalemia, hyponatremia, alkalosis, or arrhythmias but not often does so, and, since the incidence of toxic effects is small, it is ideal for long-term usage.³¹ It is well not to restrict sodium too rigidly in patients taking chlorothiazide and to instruct them to take supplementary potassium or increase their intake of foods containing potassium. The usual dosage may vary from as little as 125 mg. twice a day to as high as 500 mg. or more three times daily. It should be started in small dosage in patients taking ganglionic blocking agents, in patients who have had a sympathectomy, and in patients with coronary heart disease. Because of its diuretic action, it is especially useful in patients with coexisting congestive heart failure. When the blood pressure drops, the dosage can usually be reduced and the lowered level of blood pressure maintained.

Still more recently, hydrochlorothiazide, of which Hydrodiuril and Esidrix are examples, has been introduced.³⁶ The effective dose of this drug is in the range of 1/10 that of chlorothiazide. It has been stated that there is less tendency for hypokalemia to develop with this drug than with chlorothiazide, but it does occur and must be watched for. Its use occasionally causes a rise in blood urea nitrogen, as does chlorothiazide. Another drug structurally differing only slightly from chlorothiazide, and called flumethiazide, is currently being studied and is considered of possible "potential value as an adjunct in the therapy of hypertension."³⁷

The ganglionic blocking agents are potent antihypertensive drugs. Their use requires considerable care and supervision, and, because they are so potent, they have side effects which make their routine use somewhat undesirable. Postural hypotension may cause severe disability in some users of these drugs, and they occasionally cause severe constipation, dryness of the mouth, impotence, blurred vision, and urinary retention. When they are stopped, the blood pressure has a marked tendency to "overshoot."³¹ These drugs must be started in small dosage and gradually increased and followed by careful blood pressure checks with the patient in an upright position. The dosage must be adjusted almost from day to day, and it is usually wise to teach the patient how to measure his own blood pressure so that checks can be made before each dose of medication if necessary.

Mecamylamine (Inversine) is a completely absorbed ganglionic blocking agent as contrasted to the other ganglionic blockers which are only partially absorbed from the gastrointestinal tract. Its dose is not easily adjusted, partly because the dose may vary from as little as 2.5 mg. twice a day to as much as 160 mg. per day

and because partial tolerance to the drug can develop after a few weeks.³⁸

The average daily doses of some of the ganglionic blocking drugs are as follows: hexamethonium, 500 mg. per day with a range of 500 mg. to 4 gm.; Pentolinium (Ansolysen), 100 mg. per day with a range of 60 to 500 mg.; chlorisondamine (Ecolid), 50 mg. with a range of 25 to 200 mg.; and Mecamylamine (Inversine), 20 mg. with a range of 5 to 25 mg. and occasionally more.^{31,2}

Schroeder and Perry²² concluded their clinical conference by saying that modern antihypertensive drugs, when intelligently handled by both physician and patient, give gratifying results and are the best available specific therapeutic agents. They emphasize that, even though toxicities do occur, they are not as hazardous as the toxic effects of some of the more popular drugs and are seldom severe and rarely fatal, and their use is well justified by the seriousness of the disease for which they are used. They state finally that "there are enough drugs now available to control the hypertension and promote longevity of almost any patient who wants to be treated and will submit to therapeutic inconveniences.

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Early Diagnosis of Lung Carcinoma

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LUNG CARCINOMA is indeed a problem for *every* physician. Today the No. 1 cancer in men,¹ its statistical explosion shows no sign of regression. Most important, however, is the realization that prognosis in this disease is today *far from hopeless*. The current literature is most emphatic in support of this statement. A review of the literature for the period 1946 through 1955 brought, to this author at least, an initially surprising realization of this optimism. Among the more than 13,000 cases in this personal review, over-all survival rates of 7 to 10 per cent and of 21 to 35 per cent among the resectable cases—some 35 per cent of the total—were recorded! And today the claims are even more hopeful.² Such figures, to be sure, apply directly only to those hospitals included in the surveys—some large, some small, and a few existing solely for thoracic investigations. Nonetheless, these results should conceivably be seen in any good hospital. Why, at times, they are not is the subject of this article.

In an average city, a review was made of a number of lung cancer cases seen within a ten-year period. Rapidity of diagnosis, case management, and ultimate survival were primarily investigated.

MATERIALS AND METHODS

Site of this survey is Ottawa, Canada, a relatively nonindustrial and largely civil service town. The mean population, in the ten years covered by this survey from January 1948 to December 1957, was approximately 200,000 people. Two general hospitals serve the city, the 1,000-bed Ottawa Civic Hospital and the 600-bed Ottawa General Hospital. Branch clinics of the Ontario Cancer Foundation are attached to each hospital as well.

A total of 274 lung cancer cases were diagnosed within the survey period at the 2 hospitals. This number corresponds well to that which might have been expected. National statistics¹ indicate that in a city of 200,000, some 2,780

general cancer cases should appear within a ten-year period. Of these, 10 per cent, or 278 cases, would be specifically lung cancer.

Only those cases fulfilling two important criteria could be evaluated, however. In each instance, sufficient information for our purposes had to be available, as well as sufficient medical evidence of primary lung neoplasia. It soon became apparent that only those patients actually registered at either of the cancer clinics, where much relevant data were recorded in addition to that found in the hospital charts, would meet the first criterion. Some 199 patients, or 70 per cent of the city's total hospital cases, were so registered. Among these were all patients treated by radiotherapy. Of the remaining patients, 30 per cent of the city total, only 14 per cent had received surgery, while 86 per cent, most of whom were seen before 1954, had received either chemical treatment alone or no treatment at all. Considering the low percentage of the group not registered with the cancer clinics who received any form of really curative treatment, we may conclude that this group comprised, for the most part, somewhat more advanced cases seen at a time when radiotherapy was not so widely employed. Our survey begins, therefore, with a series of cases, which, though representative of the general situation, might tend to picture things in a slightly more optimistic manner. In that part of this article dealing with Treatment and Survival, however, statistics are shown for both clinic and nonclinic cases.

Of the 199 cases initially reviewed from the cancer clinics' files, another 55 were found to be lacking—some in hospital record data, a few in definite evidences of neoplasia. These were, however, discarded at random and should not affect the original sampling.

We have, therefore, reviewed some 143 cases of lung cancer. Sixty-six per cent of these cases had been proved by microscopic examination, another 29 per cent died with definite signs of lung carcinoma, and a final 5 per cent were lost with that which was believed to be definite lung cancer, at which time they were discharged and lost to the record departments.

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RESULTS

General case picture. Age, sex incidence, and initial and presenting symptoms at the time of hospital admission were all quite similar to the already numerous reports existing in the literature on these topics. Suffice it to conclude that lung carcinoma is an especially frequent condition above age 45, and its symptoms, both initially and later, may resemble those of any other chest condition. Cough, dyspnea, chest pain, weight loss, hemoptysis, weakness, and signs of secondary deposits were all reported as early symptoms in the foregoing order of importance. Interestingly, fully 10 per cent of symptoms seen at first were referable to secondary deposits.

How many lung cancer patients show a history of previous serious lung pathology? Confining ourselves to a five-year period prior to admission, it was found that 9 per cent had had chronic bronchitis, 4 per cent had had chronic cough, and 2 per cent had, on occasion, suffered pneumonia. Little evidence was found, therefore, to suggest carcinoma as an immediate consequence of lung disease.

As a disease of later years, lung carcinoma of course need not be the sole affliction present. Often, the presence of another concurrent condition may mask the neoplastic symptoms. In 4 per cent of cases in this survey, it was for just such an additional disease that admission to the hospital was initially requested. Diagnosis of lung carcinoma in these latter cases was the reward of routine radiographic examination of all hospital admissions.

EVALUATION OF DIAGNOSTIC PROCEDURES

Using the ideal method of direct patient interview, the cancer clinics were able in some 75 per cent of cases to provide accurate information concerning the individual case pictures prior to hospitalization. In the remaining 25 per cent of cases, the information was obtained as best as possible from the hospital records. Undoubtedly, this again introduced some bias toward the optimistic side.

The term *definite diagnosis* should be understood as meaning such a degree of certainty on the part of the attending physician that, unless diagnosed too late, he would institute definite treatment for lung cancer. *Tentative diagnosis* indicates a suggestion within the records, made either by the attending physician or a consultant, which suggests lung cancer.

General picture. Definite diagnosis was reached in some 85 per cent of all cases reviewed. An additional 12 per cent were tentatively diagnosed

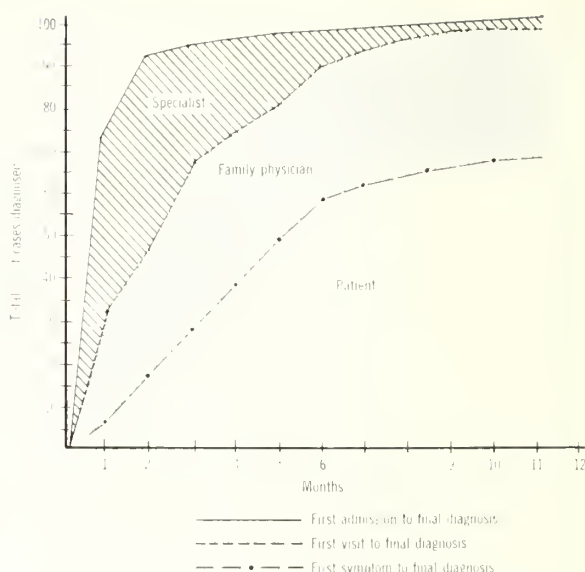


Fig. 1. Illustration of diagnostic delay showing individual responsibility. Blank area represents amount of delay due to patient, stippled area shows delay due to family physician, and vertically lined area accounts for delay due to specialist.

before death, while 3 per cent were discovered only at autopsy.

Before admission, and, in most instances, within two weeks of this time, a tentative diagnosis of lung cancer had been made in 40 per cent of cases. However, only 2 per cent were definitely diagnosed as lung carcinoma before hospitalization. In most instances, serious diagnostic procedures were not begun until patients were hospitalized.

Actual clinical stage reached at this time in individual cases was as follows: latent, 0 per cent; silent, 5 per cent; urgent, 62 per cent; and rampant, 33 per cent.

Diagnostic delay. We may consider the delay as being of 3 parts. The first covers the time from the initial symptoms to the first medical visit and is solely the responsibility of the patient. The second part represents the medical lag from the first visit until admission to the hospital and intensive investigation. The third part represents the delay until this latter search bears fruit. In some instances, these 3 divisions might be designated as the delay of the patient, the family doctor, and the specialist.

The results of investigation of diagnostic delay are shown comparatively in figure 1.

To summarize, we remark:

1. *The specialist.* Fully 49 per cent of his cases had been diagnosed within two weeks following admission, and 69 per cent had been

TABLE 1

COMPARATIVE EVALUATION OF REDUCTION IN
DIAGNOSTIC DELAY WITHIN PAST DECADE

Author	Delay		
	Patient	Doctor	Total
Overholt ³	3.8 mo.	6.2 mo.	10 mo.
Hollingsworth ⁴	3 mo.	6 mo.	9 mo.
Mason ⁵	3.4 mo.	5 mo.	8.4 mo.
Ochsner ⁶	2.8 mo.	5.5 mo.	8.3 mo.
Gibbon ⁷	3.5 mo.	4.5 mo.	8 mo.
Present survey	3 mo.	2.7 mo.	5.7 mo.

TABLE 2

METHODS OF TREATMENT

Cases	Per cent of thoracotomies	X-ray only	Other or none
Survey: 143 cases	20% (25% resected)	52%	28%
Remaining clinic cases: 56 cases	20%	52%	28%
Nonclinic cases: 75 cases	14%	0	86%
Total: 274 cases	18%	37%	45%

For explanation of division into *clinic* and *nonclinic* cases see text under Materials and Methods. Data for *Remaining clinic cases* have been interpolated from data tabulated from the 143 survey cases.

diagnosed within a month after admission. The average delay was twenty-six days.

2. *The family physician.* He referred the patient for hospitalization within two weeks in 41 per cent of cases and within three months in 79 per cent. Average delay was seven weeks.

3. *The patient.* In 41 per cent of cases, the patients waited less than two weeks before consulting their physicians after they first noticed symptoms. In 65 per cent, the delay was less than three months and, in 85 per cent, less than six months. Average delay was three months.

Of special importance, however, is the answer to 2 questions. Has there been an improvement in diagnostic delay within the past decade? Who has made the least improvement?

To reply to the first question, representative figures from the previous decade must be examined. These have been recorded in table 1.³⁻⁷

Even allowing for the slightly optimistic bias in our material, as described before, it still definitely seems as if a real reduction in diagnostic delay has been achieved by the physician. The delay attributable to the patient has meanwhile remained stable. It is the patient himself who now appears to be the greatest impediment toward earlier diagnosis.

TABLE 3

AROUSAL OF INITIAL SYMPTOMS OF
LUNG CARCINOMA

Radiography alone or with other methods	87%
Radiography alone	43%
X-ray, history, and physical combined	12%
History and physical alone	10%
Routine x-ray alone	8%
History alone	3%
Other (biopsy, pleural fluid, etc.)	24%

TREATMENT

All of our patients fell within 1 of 3 groups. Some received benefit of thoracotomy, with or without subsequent resection or radiotherapy; others received radiotherapeutic treatment alone; while the remainder were managed by palliative nitrogen mustard, hormones, or no treatment at all. The findings are shown in table 2.

Note the specific number of cases which underwent thoracotomy. *Only 20 per cent of our 143 cases and 18 per cent of all the cases were so investigated.* The resection rate nonetheless was no higher than might be expected from the literature. It should be pointed out that within the past three years, and especially in that time following the survey period, the number of thoracotomies carried out has increased considerably at both hospitals. A preliminary rise in survival rates has been noted as well.

SURVIVAL

We were fortunate in being able to trace 136 of the 143 patients presently under discussion. Fifteen patients had survived twelve months after admission. But, of these, only 5 patients, in 1 of whom cancer had never been microscopically proved, reached fifteen months' survival. The two-year survival in proved malignancies was 2.7 per cent, or 4 cases. The five-year survival was less than 1 per cent.

There were, of course, those patients—the nonclinic patients illustrated in table 3—who underwent thoracotomy but could not be included among the 143 whose cases were actually reviewed. Their survival rate was unfortunately not available; yet, even had it been, in actuality, probably a maximum of 35 per cent of these patients would have survived. The overall survival rate for the ten-year period in Ottawa would still be less than 4 per cent. All in all, the survival rate is, therefore, much less than might have been expected.

TABLE 4
DEFINITE DIAGNOSIS OF LUNG CARCINOMA

X-ray	34%
Bronchoscopy	16%
Thoracotomy	11%
Lymph node biopsy	9%
Sputum cytology	9%
Pleural fluid cytology	6%
Other	15%

TABLE 5
GENERAL ACCURACY OF INDIVIDUAL DIAGNOSTIC PROCEDURES

Test	Accuracy in proved cases of lung cancer
X-ray (all types)	98% showed some abnormality
Sputum cytology	15% definitely positive (this figure rose to 36% after 1955 at Ottawa Civic Hospital)
Bronchial aspirate cytology	20% positive
Pleural fluid cytology	36% positive
Bronchoscopy	49% positive
Bronchial biopsy	60% positive (this procedure employed only if definite lesion was seen by bronchoscopy)

It is useful to note that of the 15 patients who survived at least one year, cancer had been discovered in 7 by routine hospital or outside radiographic survey. Moreover, of the 4 proved cases of carcinoma of the lung in those whose survival exceeded fifteen months, all had had thoracotomy. Pneumonectomy was carried out in 3, while the fourth was diagnosed as inoperable and palliative radiotherapy was given. This latter patient is still alive thirty months later.

We have in the foregoing an outline of our problem. The solution to this problem must lie in the next section.

CASE MANAGEMENT

Serious diagnostic efforts, we find, are almost entirely confined to the time following hospitalization.

The findings concerning the methods of diagnosis, correct or incorrect, are reviewed in tables 3 and 4.

Apparently, thoracotomy was not done until the physicians were already rather certain of the diagnosis, and, while done in 20 per cent of cases, it accounted for only 11 per cent of the cases with a definite diagnosis of cancer.

A false diagnosis was entertained for some

time in at least 66 per cent of cases. What is especially important, however, is that, in fully 75 per cent of such cases, the only investigations to that point had been a history, physical examination, and x-ray. In other words, when cytology, bronchoscopy, or thoracotomy was employed, the diagnosis was missed in only 25 per cent of cases. One false diagnosis was made after thoracotomy. In this case, thoracotomy without biopsy was done. The lesion was described as "fibrosis of the lung," and the chest was closed. This diagnosis was corrected at autopsy.

An evaluation of the general accuracy of the individual diagnostic procedures is presented in table 5.

The striking increase in accuracy of cytologic analysis of sputum samples recorded after 1955 at the Ottawa Civic Hospital was achieved in several ways.

1. A Papanicolaou trained cytologist was hired to direct all preparation and initial screening of material. The pathologists, heretofore buried beneath stacks of ill-prepared slides, now found their time much more productively employed in the examination of only those slides which had been directed to them by the cytologist. Extension of such a department to act as a central agency for a number of smaller institutions, the cost of which could be borne proportionately by each, suggests itself as well.

2. An intensive campaign among hospital physicians to emphasize the proper collection methods for cytologic material was carried out.

3. The use, by a few physicians, of aerosols to increase deep sputum yield.

It is table 6, however, which is intended as the pièce de résistance for this particular survey. How thorough was individual case investigation?

Of the original 143 patients, the cases of 3 had already been diagnosed by admission, and for 11 others we were lacking complete information for this section. Therefore, 129 cases remained and have been reviewed regarding the relation of clinical suspicions, actual investigations, and time of definite diagnosis.

No less than 127 patients showed some sign of definite lung pathology or respiratory symptoms on admission. All had been investigated with at least a history and a physical examination, and 91 had been examined radiographically shortly after admission. Within the first three days, investigations other than histories or physical examinations were carried out in 103 cases, and 17 diagnoses of lung carcinoma were made.

But, the situation, it may be seen, changes rapidly after the fourth day. The percentage of

cases investigated falls sharply from 50 per cent between the fourth to sixth days after admission to only 22 per cent between the twenty-fifth to thirtieth days. Moreover, many of these investigations comprised but a few anteroposterior flat plates. It is to be noted, however, that even as the investigation rate fell, the ratio of cases diagnosed to cases investigated remained at a constant 30 to 35 per cent.

Generally, within the first fourteen days after admission, 53 per cent of the patients received no specific investigation for carcinoma of the lung other than physical examination and radiography.

And, while 62 per cent of all cases had been diagnosed by the thirtieth day, *only 8.5 per cent had undergone thoracotomy*. Here, we suggest, is the key to the solution of our problem posed by the low survival.

DISCUSSION AND CONCLUSIONS

The preceding suggestion is most certainly not novel. The literature within the past decade has been strongly in favor of earlier thoracotomy. A most persuasive work in this regard has been presented by Gibbon and associates.⁸ Basing

their conclusions on a group of 17 reports dealing with survival in cases of lung carcinoma, they demonstrate that a definite correlation is to be found between the ratio of thoracotomies performed and the rate of immediate salvage. When 54 per cent of patients in a series underwent thoracotomy, the salvage rate was 24 per cent; when the procedure was performed in 36 per cent, the salvage rate was 14 per cent; thoracotomies performed in 22 per cent resulted in 5 per cent salvage; and, when done in 13 per cent, salvage was only 3 per cent. These findings are in general agreement with our results.

Overholt and Atwell⁹ have estimated that an analysis of lung lesions unverified prior to exploration would prove some 40 to 45 per cent to be carcinoma. Paulson,¹⁰ in a series of 108 such cases, obtained a figure of 34 per cent. As Overholt has gone on to emphasize, in other abnormalities producing such a suspicious radiographic shadow, the lesions, while not neoplastic, threaten the well-being of the patient and are better removed.

Two equally important and synergistic factors must therefore be considered if maximum survival rates are to be achieved. These factors are

TABLE 6
DIAGNOSTIC PROCEDURES EMPLOYED IN SUSPECTED CASES OF LUNG PATHOLOGY

TOTAL - 143 NO INFO- 11 DIAGNOSED- 3 UNDIAG. -129	DAY 1 - 3			DAY 4 - 5			DAY 7-10			DAY 11-14			DAY 15 -20			DAY 21-25			DAY 26-30		
	TO BE DIAGNOSED- 129			TO BE DIAG.-109			TO BE DIAG. -96			TO BE DIAG.- 81			TO BE DIAG.- 73			TO BE DIAG. - 58			TO BE DIAG. - 49		
	SUSP.- 127 INVEST.-103			SUSP.-107 INVEST.-46			SUSP.- 96 INVEST.-33			SUSP.- 81 INVEST.-17			SUSP. -73 INVEST.-30			SUSP. - 58 INVEST.-19			SUSP.- 49 INVEST.-13		
	DIAG.- 17 DEAD - 1 REMAIN -109			DIAG.-13 DEAD - 0 REMAIN- 96			DIAG. -14 DEAD - 1 REMAIN -81			DIAG. - 6 DEAD - 2 REMAIN -73			DIAG.- 13 DEAD - 2 REMAIN-58			DIAG. - 10 DEAD - 1 REMAIN -49			DIAG. - 5 DEAD - 2 REMAIN- 42		
	P	NEG	D	P	NEG	D	P	NEG	D	P	NEG	D	P	NEG	D	P	NEG	D	P	NEG	D
MINIAT. X-RAY	15												3								
FLAT PLATE	89	2	6	27	3	18	2	7	1	3	20	4	12	2	6	3					
FLUOROSCOPY	3			3	2	2	1	1													
BRONCHOGRAPHY	3			2						1			2	2	3	3					
TOMOGRAPHY				2						1	1	1			1	1					
SPUTUM CYTOL.	2	2	12	1	3	12	3	1	7	1	2	3	1	1	3	1	1	1	6	1	3
BRONCH. ASP. CYT.	3	3	1	1	3	1	1	2	2		1		2	1		1	3				
PL. FLUID CYT.	2		1	1				1	2		1				1						
LYMPH NODE BIOPSY	5			1	2	2	2	4	2	3	1		1	2		1			1		1
BRONCHIAL BIOPSY	1				1	1		2			1		1	1		1	1	1			
BIOPSY 2NDARY				2		1	2														
BRONCHOECOPY	11	5	8	3	4	2	6	3	5	2			2	2	2	5	2	3	1	1	1
THORACOTOMY							2		2		1		3		3						

Results are tabulated generally as P for *positive*, Neg for *negative*; in the case of cytological tests the negative column is further subdivided into a column on the left for *abnormal* and a column on the right for *negative* results. Those tests resulting in a definite diagnosis of lung cancer are noted in the column labelled D.

early diagnosis and the intelligent use of thoracotomy as a method of diagnosis. One without the other would be much less effective.

That a reduction in total diagnostic delay has been achieved over the past decade would be suggested by this investigation. It has, however, been the victory of the physician; the patient himself remains an important factor retarding further reduction. How can this be overcome? The subject has already received more than a little thought. One thing is important and that is that the physician himself is in a good position to help ameliorate the situation. His most important tool in this respect is the x-ray. The mass radiographic survey, the routine hospital admission x-ray, and, by far the most important, the routine office radiographic or fluoroscopic examination at least once every year for each patient are methods that should be utilized to their most efficient extent by the profession.

In conclusion, from evaluation of the data in table 6, a major factor in the early diagnosis of lung carcinoma is a dynamic application of specific methods for the detection of this condition in suspicious cases.

The nature of this illness demands action. *Every* patient, especially if over age 45, must be managed in such a manner that if carcinoma does exist, it will be discovered and treated promptly.

SUMMARY

Five-year cure rates of 7 to 10 per cent of all cases, and 21 to 35 per cent of resectable cases have been reported in the literature. Why some hospitals fail to obtain such results is the subject of this report. A review of 143 lung cancer

cases over a ten-year period has been made. Primarily, a diagnostic delay of three months was found to be attributable to the physician and, of another three months, to the patient. The former figure is a definite improvement over figures registered in the previous decade. The latter has remained unchanged. Yet, even with this favorable rate, the five-year cure rate was found to be quite low in comparison to that which might have been expected. It is suggested that because early diagnostic thoracotomy was only infrequently carried out in the cases reviewed, the advantages of earlier diagnoses were obviated.

The author extends appreciation to Dr. J. S. Campbell of the Pathology Department, University of Ottawa, for his advice and encouragement. Thanks is offered to Dr. A. F. Crook and Dr. T. G. Stodart of the Ontario Cancer Foundation clinics for allowing generous examination of case records.

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PAIN in the right upper quadrant is the most frequent symptom of carcinoma of the gallbladder. Anemia, leukocytosis, and impaired liver function may also be seen. When possible, resection of the gallbladder and tumor is performed, and right hepatic lobectomy is advisable to avoid recurrence or metastasis. Early cholecystectomy for cholelithiasis may reduce the incidence of gallbladder carcinoma.

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Chronic Idiopathic Pericardial Effusion: Management by Early Pericardiectomy

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UNTIL VERY RECENTLY, chronic pericardial effusion has been treated expectantly, needle aspiration of the accumulated fluid usually being carried out only for diagnostic purposes or to relieve actual or impending cardiac tamponade. In 1951, for cogent reasons which will be mentioned later, Holman and Willett¹ advocated early pericardiectomy as the treatment of choice in patients with tuberculous pericardial effusion and cardiac compression. Within the past few years, several articles have appeared in the literature²⁻⁵ recommending the extension of this therapeutic principle to the management of patients with chronic pericardial effusion of unknown etiology. The small experience reported thus far suggests that this form of treatment has considerable merit.

The purpose of this paper is to record another case of successful treatment of chronic idiopathic pericardial effusion by early pericardial resection and to review briefly the etiology, diagnosis, management, and prognosis of this condition. The case to be described concerns a patient who had cardiac tamponade due to a frankly bloody pericardial effusion of record or near record proportions. Four thousand cubic centimeters of grossly hemorrhagic fluid was aspirated from the pericardial cavity at the time of surgery after a total of 1,000 cc. had been removed by pericardiocentesis on 2 occasions shortly prior to operation. Although death appeared imminent when he went to surgery, the patient's relief was immediate and dramatic and his post-operative course has been most gratifying.

CASE REPORT

The patient, a 44-year-old aircraft assembler, was admitted to Mercy Hospital on August 29, 1958, complaining chiefly of severe dyspnea and chest pain on the left.

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Although he was somewhat vague relative to the onset of his symptoms, it appeared that his present illness had begun insidiously about one year previous. He did not consult a physician until June 1958, when he complained of increasing shortness of breath. Chest roentgenograms taken at that time and later reviewed by us revealed an enlargement of the cardiac silhouette (figure 1). He was able to continue at work, however, until three weeks before his present hospitalization. During this interval his symptoms gradually increased from mild chest discomfort, especially on turning his head to the right, to a feeling of bandlike constriction of the chest. Concomitantly, his shortness of breath increased until he had incapacitating dyspnea and orthopnea. The chest pain did not radiate. He had had a productive cough recently, raising yellowish white mucoid sputum which never contained blood. During the few weeks immediately preceding this hospitalization, he had "sweats" of increasing frequency and severity.

Just prior to being seen by us the patient was studied at another hospital, where 2 pericardiocenteses were

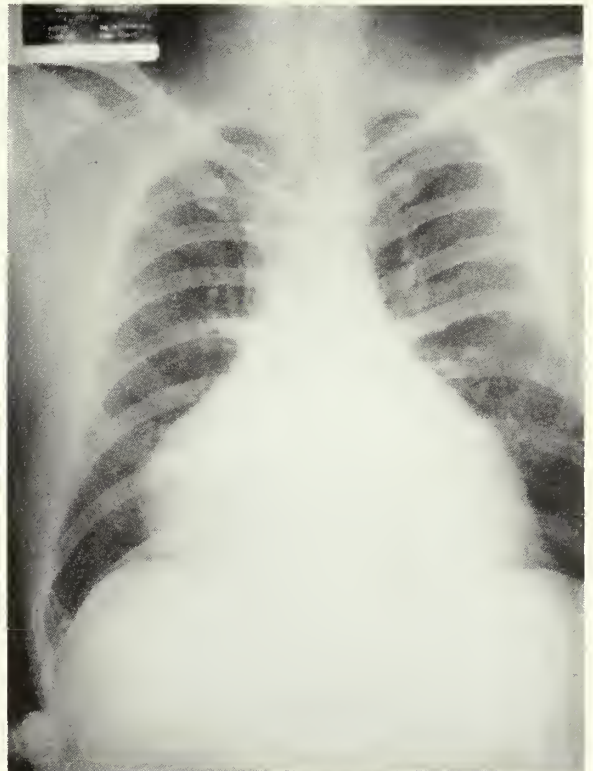


Fig. 1. Enlargement of cardiac silhouette

done. The first yielded only a few cubic centimeters of yellowish amber fluid, while at the second pericardial tap 500 cc. of grossly bloody pericardial fluid which did not clot was aspirated. Although fluid was still being obtained freely at the second pericardiocentesis, the procedure was terminated because of the frankly hemorrhagic character of the fluid being removed. Smears of this fluid provided no diagnostic information.

The patient's history disclosed pneumonia during childhood, as well as measles, mumps, and chickenpox, but he denied having had other diseases, including rheumatic fever and chorea. The patient's appetite had been good until about one week before his present hospitalization. There had been about a 10-lb. weight loss in the preceding ten days.

The patient recalled an attack of migratory arthritis which occurred in 1936 or 1937. His symptoms disappeared within a few days of their onset and did not recur. He was vague about this episode, since he had attached little significance to it at the time and did not consult a physician.

Family history revealed nothing of importance. His mother died in childbirth and his father is living and well. There are no siblings.

The patient was a pale Italian-American man in obvious distress with dyspnea, orthopnea, and a hacking, slightly productive cough. His respirations were rapid and heaving in character. Apprehension was apparent, and the patient sat upright in his bed or bent slightly forward. He complained of chest pain on the left related to respiration and aggravated by deep breathing or coughing but described also a steady precordial pain which felt "as if his chest were in a vise." He also men-

tioned profuse sweating, chills, weakness, and a feeling of depression.

The pulse rate was 120 per minute and blood pressure 130/90 mm. Hg. The accuracy of this initial reading is questionable. All subsequent blood pressure determinations were at a considerably lower level and the pulse pressure continued to be narrow. A friction rub was not heard either then or thereafter. While the patient was in a semireclining position, the left border of the heart was percussed at the anterior axillary line and the right border just beyond the right nipple line. A fairly large area of dullness on percussion was noted below the angle of the left scapula. This area encompassed the lower third of the left posterior chest wall (Ewart's sign). There was some bronchophony and there were a few medium moist basal rales. The pulse was weak and rapid and disappeared completely during inspiration (paradoxical pulse). The heart sounds were muffled. Pressure over the liver resulted in engorgement of the neck veins, elicited marked tenderness, and provoked extreme apprehension.

Laboratory Data. Admission red blood cell count was 3.83 million; hemoglobin, 11.5 gm.; and white blood cell count, 9,700, with 5 per cent stab cells, 74 per cent segmented cells, 19 per cent lymphocytes, and 2 per cent monocytes. The urine contained a trace of albumin but was otherwise negative. The specific gravity was 1.025. The chest x-ray showed a tremendous cardiac shadow (figure 2), measuring 26 cm. in transverse diameter. The purified protein derivative skin test was negative in the first and second strengths. The C-reactive protein reaction was 2+. The antistreptolysin titer, determined after pericardiectomy, revealed a negative RA test and

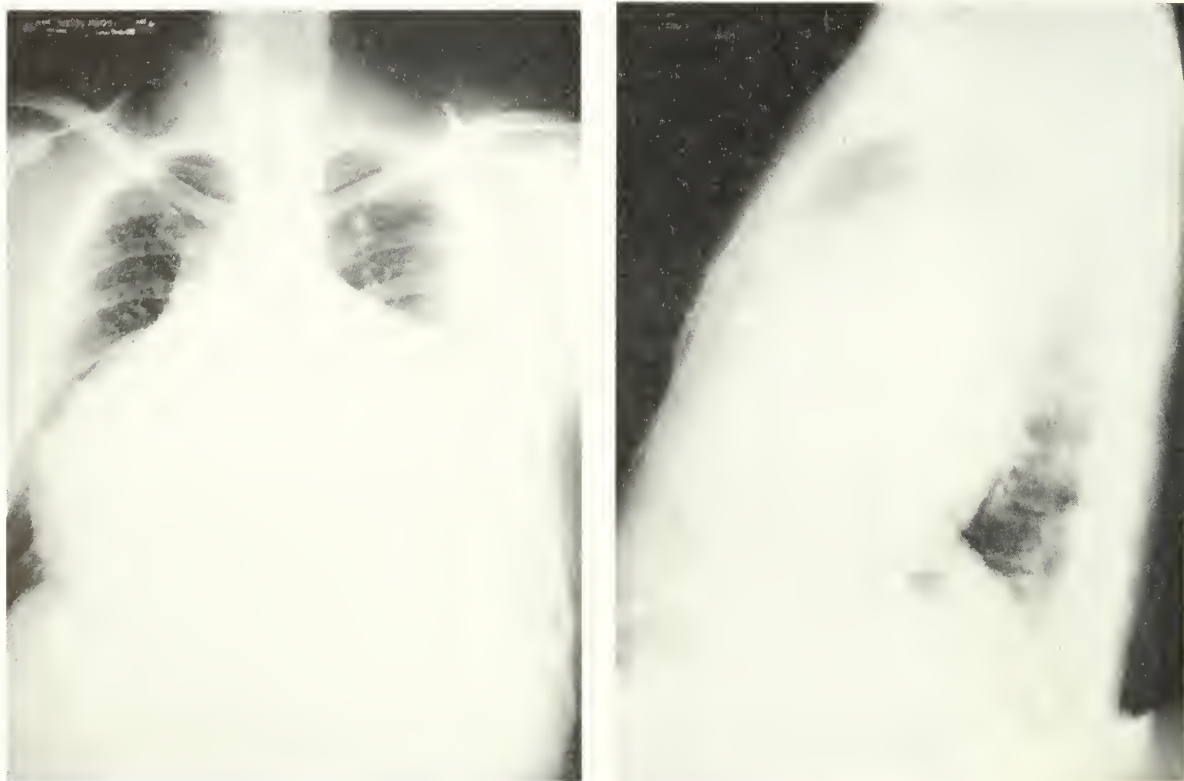


Fig. 2. Large cardiac shadow, measuring 26 cm. in transverse diameter

antistreptolysin O titer of 250 Todd units. Five days after the original determination, 500 Todd units were recorded on this test, and ten days later the value was 333 Todd units. An L.E. cell preparation was negative. The basal metabolic rate was -2 . Smears and cultures of the fluid aspirated from the pericardial cavity revealed no growth. A blood culture was also negative. The pericardial fluid contained approximately 5 gm. of hemoglobin per 100 cc. Cell block of the pericardial fluid sediment showed no malignant cells.

Two days prior to surgery the venous pressure in the left arm was 300 mm. H₂O. His arm-to-lung circulation time (ether) was 16 sec., in comparison to the normal 3 to 8 sec. The arm-to-tongue circulation time could not be determined because of the patient's inability to cooperate adequately. One week postoperatively his venous pressure was 110 mm. H₂O and the arm-to-lung circulation time, 10 sec.

The Electrocardiogram. The electrocardiographic findings in this case were of some interest. Tracings before pericardiectomy revealed sinus tachycardia with low voltage in all leads (figure 3). Postoperatively the deflection of all complexes almost doubled (figure 4). Al-

though at no time was electrical alternans, as discussed by McGregor and Baskind,⁶ found in this patient's electrocardiograms, definite fluctuations in voltage were seen. This phenomenon was best demonstrated in lead V₃ (figure 3). One could not definitely rule out respiratory variation as the cause of the findings, but, conversely, this fluctuation of voltage would agree with the concept of voltage variation commonly associated with pericardial effusion. Whether the voltage fluctuation could be attributed to movement of the heart within a fluid-filled pericardial sac is a moot question. It is noteworthy, however, that this voltage variation promptly disappeared after pericardiectomy (figure 4). Simultaneous electrical alternation of both auricular and ventricular complexes, as described by others,⁶ was not present in any of this patient's electrocardiograms.

Hospital Course. The patient was acutely ill on admission, with marked dyspnea, pale appearance, marked perspiration, and significant apprehension. His condition progressively worsened, with increasing dyspnea and orthopnea, extreme restlessness, and constant chest pain. His pulsus paradoxus was pronounced. On September 4, 1958, two days prior to pericardiectomy, 500 cc. of

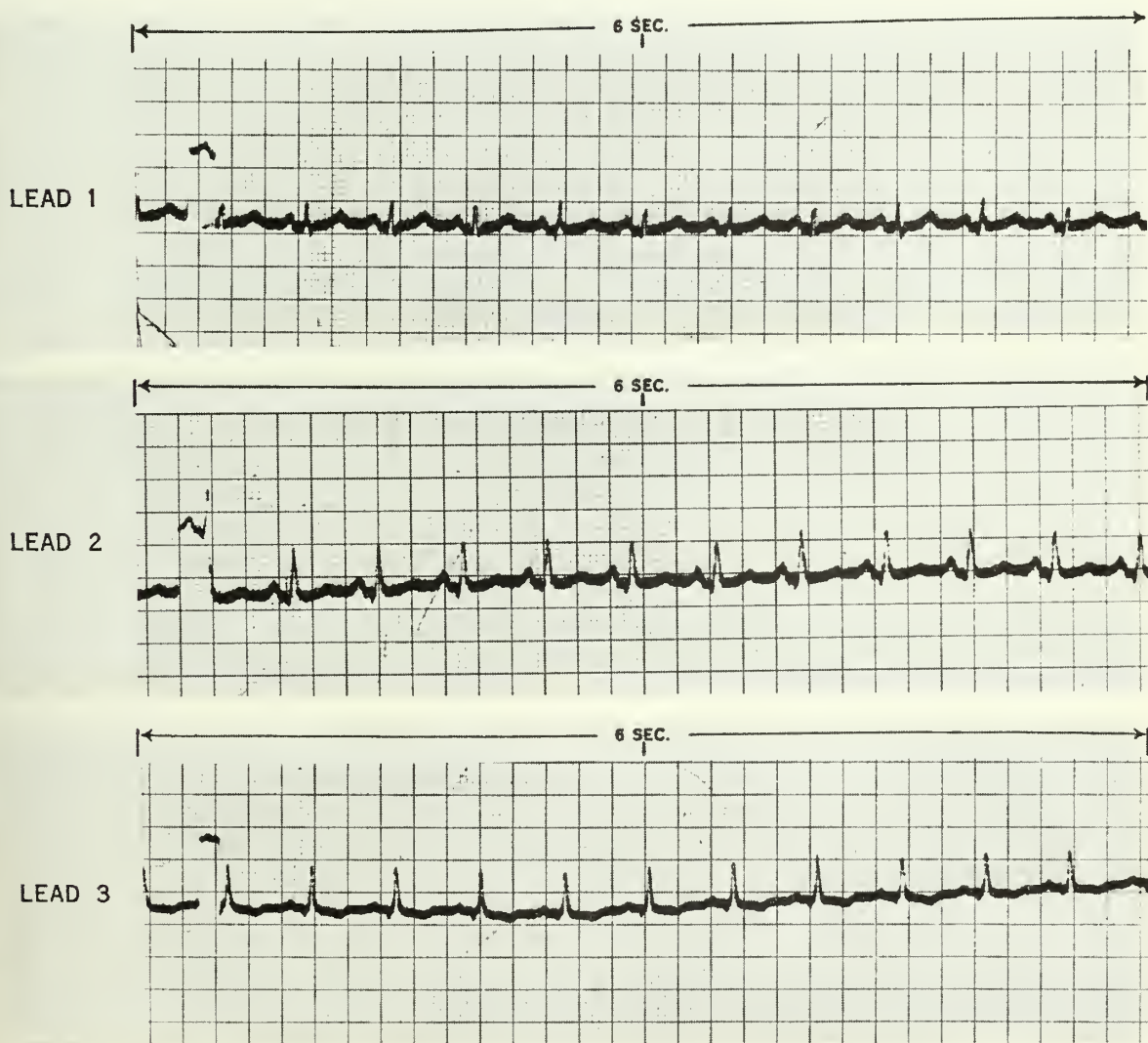


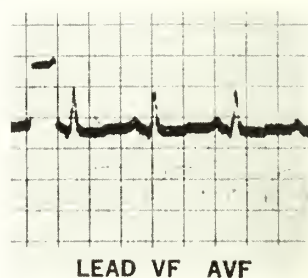
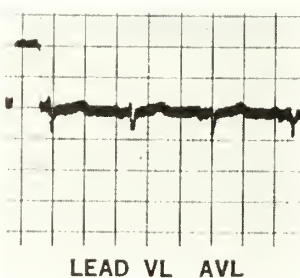
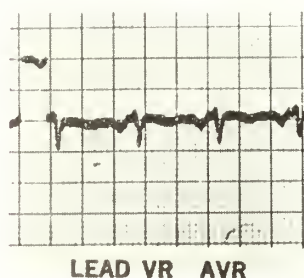
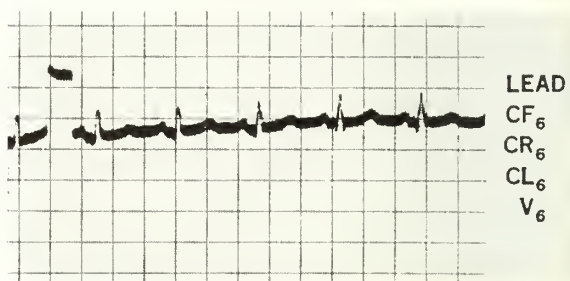
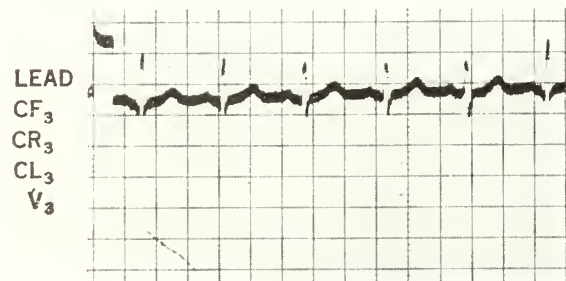
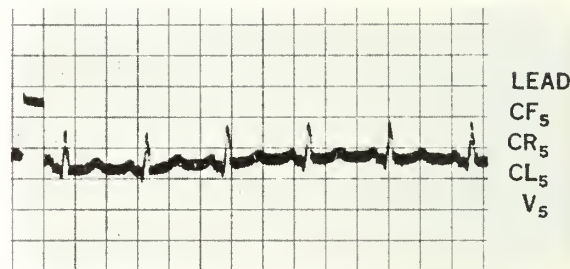
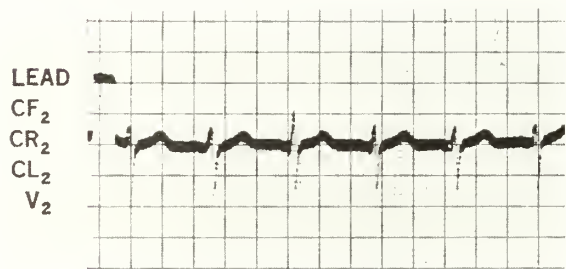
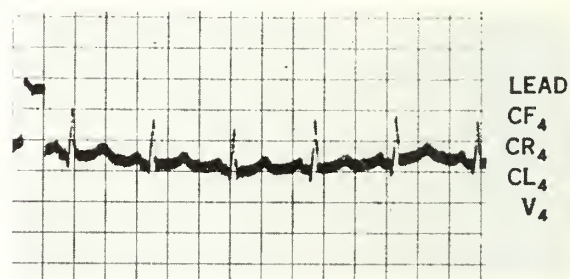
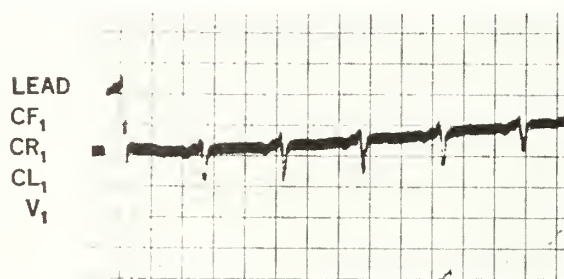
Fig. 3. Tracings showing sinus tachycardia with low voltage in all leads

grossly bloody pericardial fluid was removed at pericardiocentesis performed via the subxiphoid approach. The patient experienced some relief immediately after the tap and his paradoxical pulse became less marked. During the next forty-eight hours, however, his condition showed rapid and progressive deterioration. All of his symptoms were aggravated, and by September 6, 1958, his pulse rate had risen to 140 beats per minute, his blood pressure was 98/90 mm. Hg, and his pulse was imperceptible during 75 per cent of the respiratory cycle. At this time it was obvious that the patient was in a critical condition from cardiac tamponade and that definite relief was imperative. Pericardiectomy was elected as the procedure of choice and the operation was performed on the afternoon of September 6 with the patient under endotracheal anesthesia. At operation a huge and tensely distended pericardial sac was encountered. The pericardium was incised and 4,000 cc. of grossly bloody fluid aspirated. The patient's pulse pressure promptly increased from about 10 mm. Hg to around 40 mm. Hg, and his

blood pressure rose to 125/85 mm. Hg. Almost a total pericardiectomy was performed, an estimated 95 per cent of the pericardium being removed.

Histologic examination of the resected pericardium revealed nonspecific fibrous pericarditis. The pericardium was noted to be composed of markedly thickened and densely fibrotic tissue containing areas of vascular congestion and areas of recent interstitial hemorrhage. Foci of lymphocytic infiltration and areas of infiltration with multinucleated foreign body giant cells were also observed. There was evidence of intramural hemorrhage with cholesterol deposition and foreign body giant cell reaction. The microscopic diagnosis was "non-specific fibrinous pericarditis." A cell block of the fluid removed at operation contained no neoplastic cells. Smears of this fluid showed no organisms and cultures revealed no growth. A pericardial tissue culture was also negative.

The patient withstood the surgical procedure exceptionally well and his postoperative course was essentially uncomplicated. Remarkable clinical improvement fol-



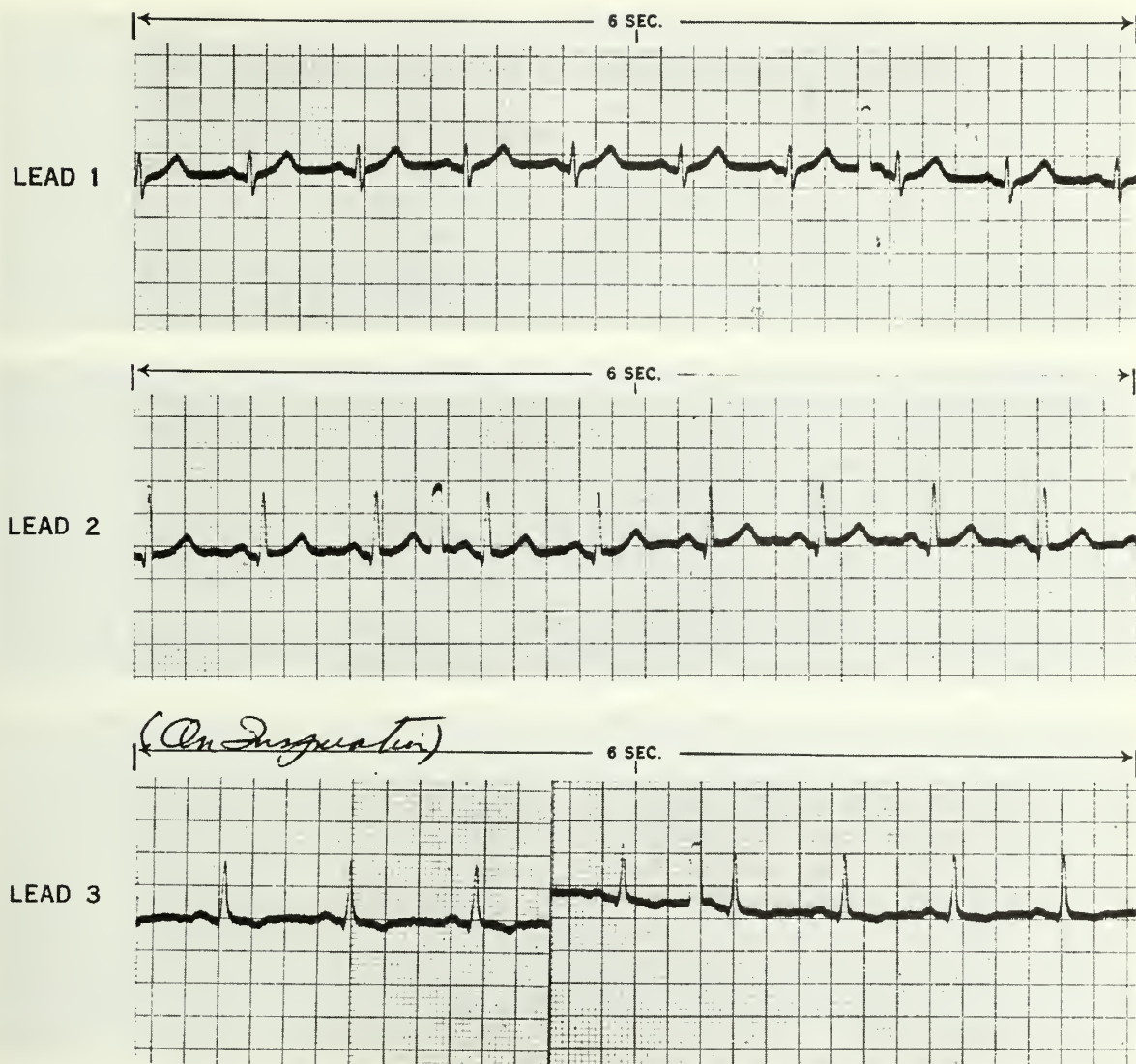


Fig. 4. Postoperative doubling of all complexes and disappearance of voltage variation

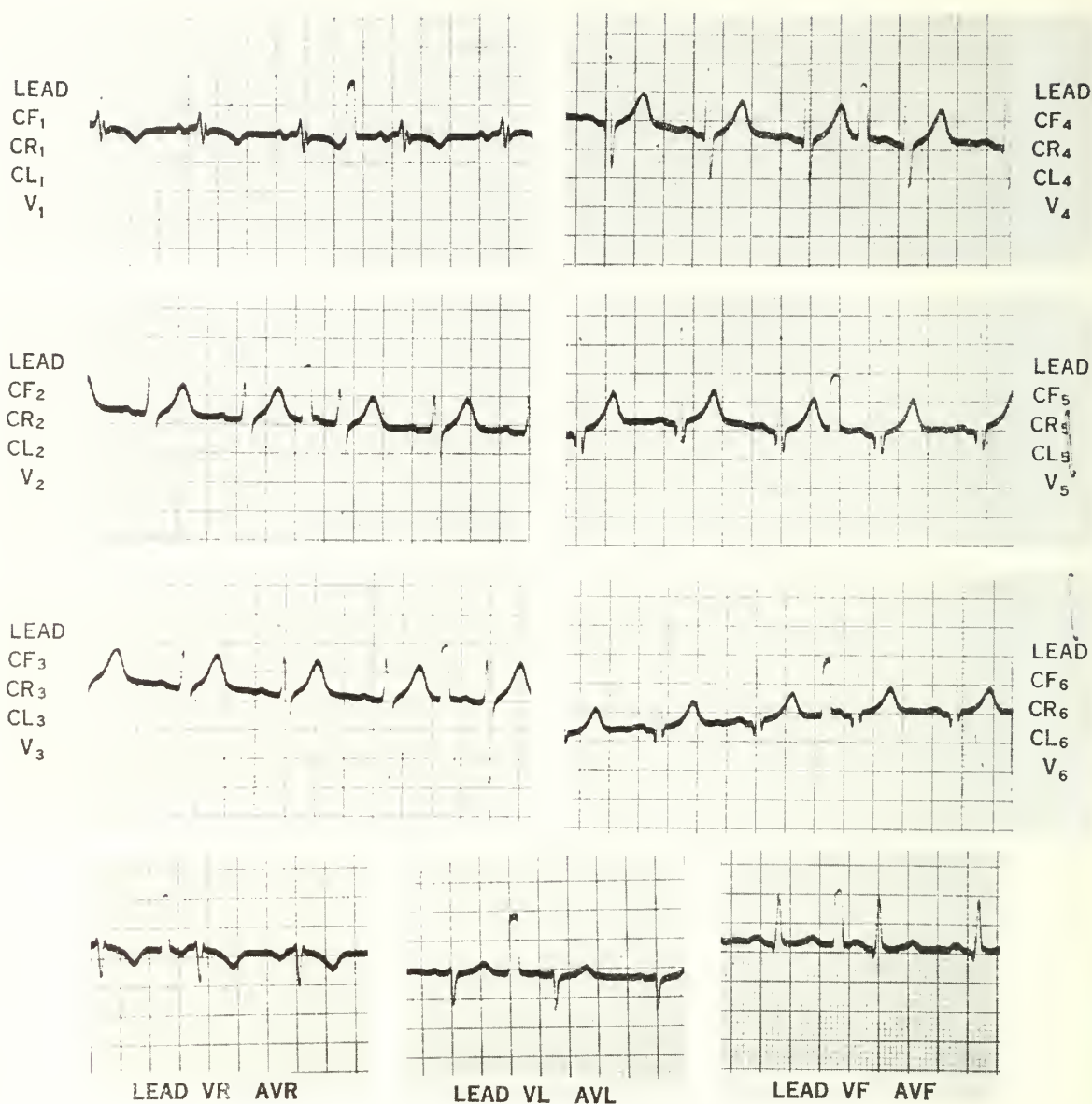
lowed the pericardial resection. There was prompt and complete disappearance of the paradoxical pulse, loss of apprehension, cessation of excess sweating, and return of respiration to normal. He was discharged on the seventeenth postoperative day and his subsequent course has been excellent. Follow-up examination eight weeks after pericardiectomy revealed a healthy-appearing man who had no complaints. His blood pressure, pulse pressure, and respiratory rate were normal. Mild tachycardia persisted. The heart sounds were normal and the chest was clear on percussion and auscultation. His chest x-ray was negative (figure 5). The patient continued to feel well and has been allowed to return to work. He remains well fourteen months postoperatively.

Comment. This case presents several features of unusual interest. The amount of fluid aspirated from the pericardial cavity at the time of surgery (4,000 cc.) appears to represent the largest amount ever removed from a living patient on a single occasion. This patient provides another example of a chronic pericardial effusion so massive that it posed a distinct threat to life as

the result of cardiac tamponade. The fact that grossly bloody effusions do not necessarily indicate the presence of a malignant lesion is again illustrated. Finally, the patient's clinical course demonstrates the prompt and apparently permanent relief that results from pericardiectomy performed for chronic idiopathic pericardial effusion with cardiac compression.

DISCUSSION

Etiology. Tuberculosis was long popularly believed to be the most common cause of chronic pericardial effusion and its late sequela, chronic constrictive pericarditis. While this is possibly true, conclusive proof to this effect has not been offered. On the contrary, intensive histologic and bacteriologic studies in relatively large series of patients with chronic pericardial effusion or constrictive pericarditis have failed to establish a precise etiology in the majority of cases. Most



such lesions, therefore, must be classed as idiopathic in origin. For example, in a recent report by Steinberg and associates⁷ on 30 patients with chronic pericardial effusion, 4 cases were of tuberculous etiology, 4 were due to rheumatic fever, and 2 resulted from myxedema, while 1 case each was caused by metastatic malignancy, lupus erythematosus, and radiation fibrosis. The remaining 17 cases (57 per cent) fell into the idiopathic category. Among 61 instances of chronic adhesive pericarditis, Brooks and Lipencott⁸ stated that tuberculosis was the *apparent* cause in 17 (28 per cent) of the patients. In none of these, however, was the diagnosis confirmed either histologically or bacteriologically.

A wide variety of inflammatory or infectious processes, metabolic disorders, neoplastic lesions, nutritional disturbances, and physical agents have been implicated as occasional causes of serous or nontraumatic bloody effusions within the pericardial cavity. Among these may be mentioned spontaneous bleeding associated with anticoagulant therapy, intrapericardial leakage of an aortic aneurysm, myocardial infarction with or without overt congestive heart failure, rupture of a coronary vessel, serum sickness, polyserositis from any cause, various acute and chronic infections, amyloidosis, myxedema, uremia, scleroderma, and other collagen disorders, such as periarteritis nodosa. Acute cardiac tamponade, most often due to intrapericardial hem-

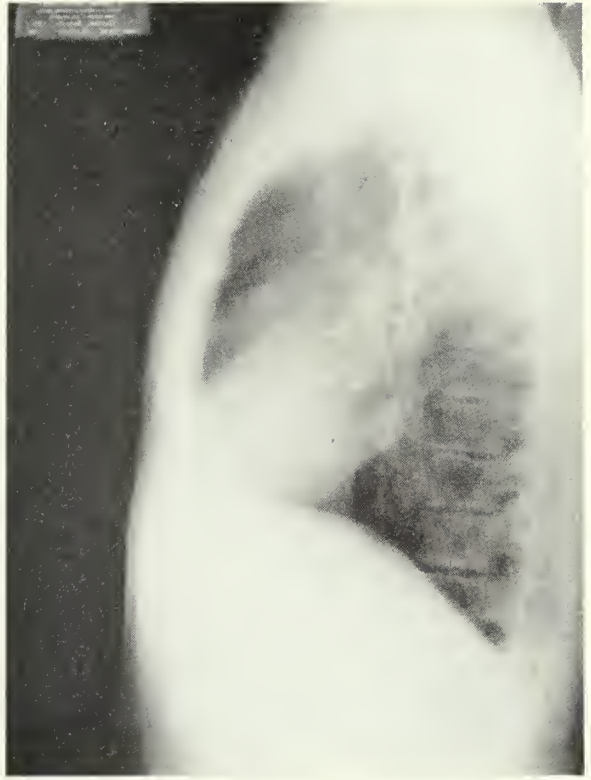


Fig. 5. Normal chest x-ray eight weeks after pericardiectomy

orrhage resulting from stab or gunshot wounds, poses a different problem and is not being considered in this discussion.

Diagnosis. The diagnosis of a relatively large pericardial effusion with some degree of cardiac tamponade usually is not considered difficult. The patient is commonly dyspneic and orthopneic, often apprehensive; the neck veins are frequently distended, and the face may be dusky. A low blood pressure, narrow pulse pressure, faint heart sounds, prolonged circulation time, increased venous pressure, and an enlarged area of cardiac dullness are characteristically present in this disorder. The pulse rate is rapid and the pulse frequently paradoxical, becoming weaker or disappearing altogether at the wrist on inspiration.

The electrocardiogram shows low voltage in all leads; the T waves may be flattened or inverted and the P waves broad and notched. McGregor and Baskind⁶ have described an electrical alternans or a less regular fluctuation of voltage, not due to respiration, which they believe to be of some diagnostic value in pericardial effusion. They state that simultaneous alternation of auricular and ventricular complexes has been observed so far only in cases of pericardial effusion and consider this association to be a unique one.

Radiographic examination in the typical patient with a large effusion reveals a pear-shaped or "water-bottle" configuration of the cardiac silhouette when viewed with the patient in the erect position, changing to a globular contour when observed with the patient reclining. Fluoroscopic examination and roentgen kymographic tracings show diminished or absent cardiac pulsations. No change in heart size is noted when the Valsalva maneuver is performed.

While the foregoing findings in combination are highly suggestive of a pericardial effusion, they are not pathognomonic. Massive cardiac dilation may present the same picture. Therefore, even the classic signs of a pericardial effusion do not permit one to arrive at a clinical diagnosis in all cases with complete confidence in its accuracy. Furthermore, any one, or many, of the expected findings may be either absent in the individual case or contrary to the customary pattern.

Since x-rays demonstrate no difference in density between blood, water, and soft tissue, routine chest roentgenograms do not permit the radiologist to distinguish with certainty between a markedly dilated heart and one of normal size surrounded by fluid. Several radiographic methods have been suggested whereby a positive differentiation between the 2 may be made. These

include x-ray visualization of the subepicardial fat,⁹ films of the heart after the intravenous injection of carbon dioxide,¹⁰ and angiocardigrams.⁷ The first of these technics is presently in the experimental stage, but it appears that it may be useful when perfected, particularly when equipment for rapidly exposed laminagrams becomes available. In the few clinics where it has been employed, the carbon dioxide method has proved to be safe and satisfactory, but it has not been widely used.

The greatest experience has been had with angiocardigraphic studies in this condition, and angiocardigrams have proved completely reliable in distinguishing cardiac enlargement from pericardial effusion. This subject has recently been thoroughly discussed in an article by Steinberg and his associates.⁷ The safety of this method is emphasized by their experience. They report 1 death in over 3,000 patients who have had angiocardigrams. These authors mention the recent reports in the literature recommending surgical treatment of pericardial effusions and biopsy of the pericardium for diagnosis. They emphasize the importance of an accurate diagnosis of pericardial effusion if surgery is to be undertaken and express the opinion that angiocardigraphy is safer than blind-needle aspiration for this purpose.

Two other diagnostic methods may be mentioned. These include x-rays of the heart with a cardiac catheter in place within the cardiac chambers and chest roentgenograms after pericardial aspiration with partial replacement of the fluid by air. The range of thickness of the right ventricular musculature in normal individuals and in various disease states has been established quite well. This knowledge makes it possible to determine with reasonable certainty the presence of a pericardial effusion by taking films with an opaque catheter in the right ventricle. However, this involves a procedure of greater magnitude and an increased risk in comparison with angiocardigraphy, so that the latter is the preferred diagnostic technic.

Air-contrast studies constitute the time-honored and certainly the most widely used method of investigation of pericardial effusions. Although the hazards of pericardiocentesis have been mentioned and will be discussed in more detail later, it is a procedure which we have carried out on numerous occasions without particular trepidation. It is done with local anesthesia and the left subxiphoid approach is used routinely. Contrary to older teachings regarding cautions and limited removal of the accumulated fluid at the time of pericardiocentesis, it is our practice to remove

all of the effusion that can be aspirated easily through a large-bore needle when the first pericardial tap is done. No adverse effects have been noted. The injection of a modest amount of air into the pericardial cavity after the major portion of the fluid has been removed permits informative radiographic studies to be carried out after completion of the procedure. The films will show an intrapericardial air-fluid level, the approximate thickness of the pericardium, the size of the heart, the presence of any irregularities suggesting tumor, and the relative amount of the residual effusion.

Certain comments regarding the character of the pericardial fluid are in order. The characteristics of the effusion may vary considerably. It is clear, amber, and serous in some patients; thick, cloudy, and yellowish in others; and frankly bloody in a surprising number of instances. In reviewing 20 cases of pericardial effusion, Price and associates¹¹ found that pericardiocentesis had been done in 15 of them and the fluid was discovered at postmortem examination in the remaining 5. The effusion was a bloody one in 10 of these. Price mentions 2 other unreported cases of his knowledge in which there was hemorrhagic effusion, and he concludes that the fluid is bloody about as often as it is serous. Bloody pericardial effusion was present in 6 of the 16 cases reported by Proudfit and Effler,¹² 5 of these being idiopathic and only 1 due to malignancy. On the basis of their experience in finding loculated pockets of old blood when performing pericardiectomy for constrictive pericarditis, they believe that hemorrhagic effusions due to benign processes are more common than is generally realized. We have had a similar experience and consider this point worthy of emphasis.

In 3 of Steinberg and associates' 30 cases there was bloody effusion.⁷ One was due to myxedema; the other 2 were idiopathic. They have postulated that intrapericardial hemorrhage may be caused by severe mechanical stretching and tearing of the pericardial capillaries by massive effusions. They concluded that "since hemorrhage may thus be spontaneously produced, search for neoplastic sites or myocardial infarction and other causes of hemopericardium need, therefore, not be prolonged." In support of this thesis is the well-established fact that bloody fluid is rarely, if ever, encountered early in benign effusions when the fluid collection is still small in amount. Proudfit and Effler¹² mentioned the fact that the withdrawal of bloody fluid often causes an inexperienced operator to abandon the pericardiocentesis. The cited fre-

quency of bloody effusions¹¹ shows clearly that this timidity is not warranted.

The huge amounts of fluid which may accumulate within the pericardial cavity are indicated by several reports in the literature concerning massive effusions. Yu and associates² removed a total of 4,910 cc. at 3 pericardial taps performed on one patient within a period of a few weeks. The second thoracentesis in this patient, performed eight days after the original aspiration of 1,400 cc., yielded 1,910 cc. of serosanguineous fluid. The authors considered this to be a record at the time for a single pericardiocentesis. Shumacker and Harris,⁵ in 1956, reported the aspiration of 2,050 cc. at a single tap from a patient with chronic idiopathic pericardial effusion, from whom over 2,000 cc. had been removed by pericardiocentesis in the preceding three days. Proudfit and Effler¹² reported the aspiration of 3,500 cc. of bloody fluid from the pericardial cavity of a patient with metastatic malignant thymoma. Soloff and Zatzuehni¹³ report on a patient from whom "several liters" of fluid was aspirated. The precise amount was not stated, nor was it made clear that this was removed at a single tap.

Treatment. In their article originally advocating the treatment of active tuberculous pericarditis by pericardiectomy, Holman and Willett¹ point out that this disease is chronic and insidiously progressive, frequently characterized in its early stages by an inflammatory effusion, and culminating almost inevitably in great thickening and fibrous contraction of the diseased pericardium. Having observed the relentless course of the disease from its onset as an effusion to the late development of chronic constrictive pericarditis in 2 patients, the authors decided unequivocally that in each instance operation should have been performed at an earlier stage of the process. In 3 subsequent similar cases, early pericardiectomy was done, with a favorable outcome in all of them. These experiences led Holman and Willett¹ to recommend that "at the moment a diagnosis of cardiac compression is made in a patient with tuberculous pericarditis, an operation for decompression should be considered, regardless of whether the compression is noted in the stage of pericardial effusion or the stage of pericardial constriction."

Barker and Johnston,¹⁴ in a significant report, have called attention to the fact that, while pericarditis with effusion is considered to be an acute form of the disease, this is not invariably true. They have observed that it may follow a chronic course for many months without exhibiting any of the features of the acute process. These au-

thors recorded 3 such cases, all of them of unknown etiology. The patients were under observation for long periods of time, one for six years; another was known to have had an effusion for two years and probably had it for five, while the third patient was operated upon in the stage of the effusion seven months after it was diagnosed. Poor results were achieved by pericardiectomy in the 2 patients who were operated upon after chronic constrictive pericarditis had developed. In 1 of these, pericardial resection was carried out on 2 occasions, but in spite of this the final outcome was not satisfactory. The patient who underwent relatively early surgery showed appreciable early improvement, but this proved to be only temporary. Reexploration was done in May 1952 and he was found to have elevated pressures in the right ventricle and pulmonary artery. Lung biopsy revealed pulmonary arteriosclerosis. His downhill course continued, and he died in February 1953.¹⁵

In advocating the treatment of chronic idiopathic pericarditis with massive pericardial effusion by early pericardiectomy, Mannix and Dennis⁴ point out these disadvantages of following the usual regimen of repeated needle aspirations:

1. Pericardiocentesis has, on occasion, resulted in fatal hemorrhage.

2. Cardiac tamponade is usually relieved only temporarily, and prolonged tamponade may result in serious myocardial, hepatic, and pulmonary damage.

3. The longer the infection in the pericardial cavity persists, the greater the danger of progression to the constrictive phase of pericarditis.

Accordingly, they performed early operation in 4 of 6 patients with chronic pericardial effusion and obtained an excellent result in each of them. Surgery was delayed unavoidably in the remaining 2 patients, and both developed chronic constrictive pericarditis, requiring a more difficult and dangerous pericardial decortication. Meanwhile, they had suffered the adverse effects which accrue as the result of prolonged cardiac compression. Dock¹⁶ has consistently urged early intervention in chronic pericardial effusion in order to prevent vascular damage to the coronary system. He points out that these vessels, lying in the epicardium, are narrowed or occluded by a persistent inflammatory process and states that healing may result in permanent damage to them.

As far as we have been able to determine from a cursory review of the literature, the first recorded case in which pericardiectomy was performed for chronic idiopathic pericardial effusion is that of the third of the 3 patients reported

TABLE 1
CASES REPORTED TO DATE OF PERICARDIECTOMY FOR CHRONIC IDIOPATHIC PERICARDITIS WITH EFFUSION

<i>Author</i>	<i>Age</i>	<i>Sex</i>	<i>Year reported</i>	<i>Duration of symptoms</i>	<i>Results</i>	<i>Follow-up</i>
Barker and Johnston ¹¹	33	M	1950	7 months	Fair	Died in 1953 of cor pulmonale
Yu and associates ²	70	M	1953	1 year	Good	1 year
Rabiner and associates ³	40	M	1954	3 months	Good	1½ years
Mannix and Dennis ¹	41	M	1955	7 months	Good	5 months
Mannix and Dennis ¹	43	F	1955	4 months	Good	1½ years
Mannix and Dennis ¹	45	M	1955	4 years	Good	2 years
Shumacker and Harris ⁵	16	F	1956	7 months	Good	10 months
Shumacker and Harris ⁵	53	M	1956	2 years	Good	8 months
Present case	41	M	1959	1 year	Good	14 months

on by Barker and Johnston.¹¹ The operation was performed on December 7, 1949. Subsequent cases have been recorded in some detail by Yu and associates,² Rabiner and associates,³ and Mannix and Dennis.¹ Shumacker and Harris⁵ added 2 cases to the growing literature on the subject of early pericardiectomy for idiopathic pericardial effusion with cardiac tamponade. Both of their patients were operated upon during the stage of the effusion, and an excellent result was achieved in each case. For the reasons which have been mentioned previously, they concluded that early definitive surgery is the treatment of choice in patients with this condition. Table 1 lists the cases reported to date in which pericardial resection has been carried out for chronic idiopathic pericarditis during the stage of the effusion.

Incision and drainage of the pericardium apparently was performed by Romero¹⁷ as long ago as 1819, but after enjoying a brief vogue the method was abandoned in favor of conservative treatment. Recently Field,¹⁸ Williams and Souter,¹⁹ Silverstone,²⁰ and Prondfit and Effler¹² have advocated pericardial fenestration in the treatment of benign or malignant chronic pericardial effusion. Prondfit and Effler noted that pericardiocentesis can lead to cardiac arrhythmias, syncope, laceration of the heart and coronaries, and pneumothorax. More important, they feel, is the limitation of the method in investigation and treatment. They observe that the failure to obtain fluid does not necessarily connote its absence, that a sterile culture does not conclusively rule out a specific cause, and that mere removal of the fluid by needle aspiration does nothing to prevent its reaccumulation. The much greater absorptive powers of the pleura in comparison with the pericardium are well known. It is, therefore, not surprising that the pericar-

dial window technic has given early effective relief of pericardial effusion with tamponade in those cases in which it has been employed. The subsequent fate of these patients, however, is unknown. There are indications that some of these windows close at a later date, permitting the chronic effusion to recur. Furthermore, even when patency is maintained, the presence of a small pericardial hiatus scarcely can be expected to prevent definitely the later development of chronic constrictive pericarditis.

Pericardial fenestration does not offer the distinct advantage which is gained by early removal of the major portion of the diseased pericardium. The creation of a pericardial window, a relatively simple operation, would appear to be particularly useful in the management of chronic pericardial effusion due to malignancy. However, early pericardiectomy, in our opinion, is the preferred treatment in other selected cases of massive chronic pericardial effusion with cardiac compression. This applies to idiopathic effusions, those due to tuberculosis, and certain other cases of specific etiology. Adequate removal of the pericardium is infinitely easier and far less hazardous in the stage of effusion than pericardiectomy after chronic constriction has developed. The procedure of much greater magnitude demanded in the treatment of chronic constrictive pericarditis must be done in a patient not so fit to withstand it because of the disabling effects which result from long-continued cardiac compression. Early pericardiectomy can be performed in a better risk patient who can be rehabilitated more quickly and more completely than is possible in one who has suffered irreversible damage to the hepatic, pulmonary, and cardiovascular systems from prolonged constriction of the heart.

Prognosis. The poor prognosis for patients

with chronic constrictive pericarditis is well known. An adequately performed pericardiectomy offers much to these patients, but total rehabilitation usually cannot be anticipated. Furthermore, it is well known that surgery in this condition is associated with an appreciable risk. It appears, however, that the immediate hazard to life posed by chronic pericardial effusion with cardiac tamponade has not been emphasized adequately. It is worthy of note that, in general, the total amount of a pericardial effusion is not as important as the rapidity with which it forms. The sudden accumulation of a relatively small amount of pericardial fluid may cause life-threatening cardiac tamponade, while huge effusions which have collected over a period of many months may be tolerated surprisingly well.

Steinberg and associates⁷ state that the precipitous development of discomfort in a patient with chronic pericardial effusion should cause one to suspect intrapericardial bleeding. Price and associates¹¹ reported a case of massive sanguineous idiopathic pericardial effusion in which death resulted from its sequelae. They reviewed the literature and found reports of 4 similar cases which terminated fatally. This led them to question the "benign" nature of the disease. They concluded that idiopathic pericarditis may constitute a medical emergency due to tamponade. The case reported herein provides substantiating evidence for this opinion. It would seem, then, that when chronic pericardial effusion and cardiac compression are viewed from the standpoint of both immediate and remote effects, the prognosis must be guarded.

SUMMARY AND CONCLUSION

The etiology, diagnosis, treatment, and prognosis of chronic pericardial effusion have been discussed briefly. A case of chronic idiopathic pericardial effusion treated by early pericardiectomy has been reported. This patient had a massive bloody effusion with severe cardiac tamponade.

Four liters of fluid were removed from the pericardial cavity at the time of pericardiectomy. This appears to be by far the largest amount of pericardial fluid ever removed at one time from a living patient. The rationale of early pericardiectomy in selected patients with chronic pericardial effusion has been considered. Early pericardiectomy is the treatment of choice in patients with chronic pericardial effusion and cardiac compression due to benign disease.

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INTRACARDIAC ELECTROCARDIOGRAPHY may be a useful adjunct to cardiac catheterization. In addition to reducing exposure to radiation, the procedure permits accurate localization of the catheter tip, identifies the type of arrhythmia, and detects impending endocardial trauma and unrecognized coronary sinus catheterization.

C. A. BERTRAND, L. R. ZOHMAN, and M. H. WILLIAMS: Intracardiac electrocardiography in man. *Am. J. Med.* 26:534-542, 1959.



Henry L. Ulrich, M.D.

THOMAS A. PEPPARD, M.D.

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DR. HENRY L. ULRICH came to Minneapolis in 1902. His attention was directed to this locality by a personal letter from Miss Mabel Austin, daughter of Minnesota's governor in 1870, with whom he had been acquainted in Baltimore during his undergraduate days. Her salesmanship was such that it helped the city and the state gain a good citizen and a brilliant mind.

Dr. Ulrich, son of Peter and Catherine Ulrich, was born in Newark, New Jersey, in 1876, one of a family of six. He attended the public schools in Newark, after which he attended Rutgers University, receiving his Bachelor of Science degree in 1897. Upon his graduation, he attempted to secure a scholarship to enable him to go to Columbia University with the idea of specializing in sociology. One can note in a number of his later publications his sustained interest in the social aspects of our civilization. Failing to secure the scholarship, it was necessary that he determine his future course. This he did after much thought and discussion with the family physician and with his biology professor at Rutgers. The former suggested medicine as a career, the latter recommended his own Alma Mater, Johns Hopkins.

Entering the Baltimore school, he came under the influence of the great men who redesigned medical education at the turn of the century—William

H. Welch, William S. Halsted, and Howard A. Kelly. He was house officer under Harvey Cushing and clinical clerk under William Osler. He did special work in pathology. On graduation in 1901 in the fourth class to be graduated from Johns Hopkins, he was recommended for the position of professor of pathology at the Dallas Medical School, which is now Baylor University College of Medicine. The situation at this western university did not appeal to him, trained as he had been, and so he came to Minneapolis. He at once associated himself with the University of Minnesota College of Medicine, which had been organized in 1888, and first taught clinical microscopy in a basement room of the old dispensary at Seven Corners.

It surely was to be expected that one with Dr. Ulrich's background and training would soon make his influence felt. Early in his career, he became interested in tuberculosis. He was chairman of a committee for the establishment of a clinic at the university, and he read a paper, "A Dispensary for Tuberculosis in Minneapolis," before the Minneapolis Medical Club April 20, 1903, which was later published in the *Northwestern Lancet* 23:229, 1903. From 1903 to 1906, he gave a series of lay lectures on the prevention of tuberculosis. In 1903, he was one of the organizers of the Committee on Tuberculosis of the Associated Charities, which later became the Hennepin County Tuberculosis Society. He served as president of this group in 1920 and 1921.

In 1907, he went to England where he studied under Sir Almroth Wright. After his return, he published a number of papers from 1908 through 1918. Some of his titles were "A Talk on Opsonins and Their Practical Value," *Northwestern Lancet* 28:511, 1908; "Vaccine Therapy," *Northwestern Lancet* 39:247, 1910; "Some Notes on Hay Fever," *J.A.M.A.*

The papers in the present series of biographical sketches and those already published, a list of which follows this article, and those to appear subsequently are of interest and importance from the standpoint of medical history of the Mid-Northwest and in the delineation of the lives and characters of the men who made that history.

62:1220, 1914; and "Experimental Pollinosis," *Journal of Immunology* 3:453, 1918. He made his own extracts and tells of his experience in collecting ragweed pollen in the fields in the early morning hours. Few are now aware that he devised the commonly used modification of the heat and acetic acid test for albumin in the urine, described in his publication in the *Northwestern Lancet* 29:87, 1909. For a number of years, he did Wassermann tests in his laboratory for the State Board of Health. His bibliography, which is not entirely complete, includes a total of 37 articles appearing in local and national journals from 1903 to 1958—a span of fifty-five years.

When, in 1912, the faculty decided that the curriculum should include a short course in tropical medicine, loud cries of anguish arose not only among the "students" but also from the faculty members. The myopic vision and lack of understanding of the undergraduates did not allow them to appreciate the value of such teaching, given in the northernmost state of the Union. The faculty, too, didn't care too much for such a task, for who should or could teach such a course? Their anxiety was relieved when one of the younger members—Dr. Ulrich—accepted the responsibility for the assignment, which gave him the opportunity and the stimulus to review a subject of which he had some knowledge though little experience. Among the class members, the loudest and most vociferous dissenter, Paul White Wilson, felt impelled to circulate a petition to the dean to withdraw the requirement believed to be so wasteful of valuable time. The appeal was, of course, rejected by Dr. Frank Fairchild Westbrook, and so it came about that the class was met once weekly by Dr. Ulrich and given an adequate survey of the tropical diseases. Ironically, this same Paul Wilson, later, as a career officer in the navy, came under the influence of Dr. Stitt and became a specialist in tropical diseases and taught the subject in the Navy Medical School.

In 1917, he was appointed to head the university medical service at the Minneapolis General Hospital. The situation at that time could almost be called primitive, but in his quiet, indefatigable way, Henry Ulrich brought order out of chaos and brought the service on the medical wards up to university standards and traditions. As his associate, I walked the wards with him daily, roaming the entire hospital, the laboratory, the x-ray rooms—anywhere and everywhere a problem presented. As doctors are wont to do, we disagreed most amiably. With this association, it was natural that we should often be together at local and national medical meetings. We learned to know and talk to each other, and I shall be eternally grateful for that privilege.

I find it necessary to tarry further on the subject of General Hospital. Daily bedside clinics were held, and, on Thursdays, Dr. E. T. Bell and his staff, including Dr. James McCartney and Dr. Benjamin Clawson, held pathologic conferences at 10:30 A.M., where our successes and failures were under scrutiny of the entire personnel, and the clinicians were con-

stantly being instructed in pathology. After a brief thirty-minute luncheon, medical rounds were held from 1:00 to 2:00 P.M. These were attended by the pathology group, who were thus able to observe and better understand the problems encountered on the wards.

Shortly, the stagnant B, or city section, was discontinued and the entire medical service came under Dr. Ulrich's jurisdiction. He inaugurated a residency program. Some of the early residents included Dr. John T. Quirk, Dr. Leo G. Rigler, Dr. A. E. Cardle, Dr. Morris H. Nathanson, and Dr. Julius Jensen. The stimulating and inspiring leadership of the chief made the medical residency and internship most attractive, and the confidential reports sent to the dean's office were uniform in giving top rank to the medical service. Dr. Ulrich was always encouraging to the young doctor and helpful, generous, and quick to give due credit for work well done. He did not hesitate to be critical, usually in a most kindly way, though occasionally he might inject a barb into his criticism, always with the purpose of stimulating the young physician. He was and is intolerant of sloppy thinking. He was and is a nonconformist. On occasion, he might surprise and jolt his listeners into a different appraisal of the particular problem under discussion. On one occasion, in giving the principal address at an annual meeting of otolaryngologists, he chose not to praise their accomplishments but instead to frankly tell them that they had bartered their heritage for a mess of instruments.

In an editorial entitled "Whither Bound, Internist?", published in *Minnesota Medicine* 28:663, 1945, he remarks that the internist "is so immersed in the dialectics of his work that even his rightful position in the hospitals has disappeared unnoticed by him, due entirely to the meekness of the internist. To regain even a semblance of recognition, he must combine in an aggressive group and recapture some sort of niche in the hierarchy."

Dr. Ulrich's sense of humor is keen. He loves a good story, and those he tells carry a subtle point. He is interested in words and their meanings and often turns to his dictionary to determine or reassure himself concerning their derivations. To suit his purpose, he may coin a word, as he did in the title of one of his articles, "Straptococciosis," published in *The Journal-Lancet* 35:627, 1915. He expresses himself carefully, thoughtfully, and, usually, briefly, often turning out a neat and unusual phrase of which he is quite fond. A favorite quotation of his is from Sir Francis Bacon, "I hold every man a debtor to his profession."

Dr. Ulrich is always gracious, and, at his office or at his home, the visitor is met with a genial smile and a warm handclasp, leaving no doubt in his mind of the doctor's real and sincere pleasure in seeing him.

In World War I, he served as captain in the medical corps of the army. He was a charter member of the Minnesota Society of Internal Medicine and later served as its presiding officer. In 1926, he

served as president of the Minnesota Academy of Medicine. In 1929, he was president of the Minnesota Pathological Society. In 1936, he was president of the Hennepin County Medical Society. In 1952, he was the recipient of the St. Barnabas Bowl awarded annually to the doctor "who by reason of his professional contributions on the basis of medical research, medical achievement, or leadership has become the outstanding doctor of this or other years." He was the first Minneapolis physician to install an electrocardiograph in his office, which he did in 1919. Serving as librarian of the Hennepin County Medical Society, he was helpful in developing its library to a high degree of efficiency.

His reputation grew slowly and surely. In the "Autocrat of the Breakfast Table," Dr. Oliver Wendell Holmes compares men to fruit—pears, I believe,—those maturing early, ready to eat as plucked from the tree and those that need to be kept to be enjoyed later. Dr. Ulrich belongs to the latter group.

He advanced in academic standing and served as clinical professor of medicine at the University of Minnesota from 1927 to the time of his retirement from the university in 1944. While continually keeping abreast of advances in medical science, he would be complimented by being termed a physician of "the old school."

At the university continuation course in 1957, Dr. Ulrich gave an introductory talk on the physical examination, in which he remarked: "To develop this habit [making a thorough physical examination] and having acquired it, you will have one of the remaining joys of practice. You must have clean hands, good health, no sense of time, and a clear conscience. It is essential for your self respect and for your growth as a physician. This is the core of your developing medical personality. By sending your patient around to laboratories, you are losing your power of observation, sense of touch, and keenness of differentiation of sounds. This type of practice may be more advantageous to save time, but, in the meanwhile, you may have lost your medical soul."

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Moses Barron, M.D., by E. T. Bell, M.D. *Journal-Lancet* 79:522, 1959.

Elevison Thompson Bell, M.D., by Lemuel J. Wells, M.D. *Journal-Lancet* 80:41, 1960.

THE PSYCHOLOGIC effect of separating an infant from its mother appears to be dependent upon the child's age and stage of development. Infants less than 7 months of age do not appear to be upset by a separation, while, in older infants, extreme emotional reactions are frequently seen. Adjustment to hospitalization is rapid in the infant under 7 months, whereas older infants often exhibit fear and restlessness and are liable to develop difficulties in eating and sleeping. When a choice is permitted, as in cases of elective surgery, hospitalization before the age of 7 months is recommended.

H. R. SCHAEFER and W. M. CALLENDER: Psychologic effects of hospitalization in infancy. *Pediatrics* 24:528–539, 1959.

Book Reviews

The Surgeon and the Child

WILLIS J. POTTS, M.D., 1959. Philadelphia: W. B. Saunders Co. 255 pages. Illustrated. \$8.00.

This book is an excellent review of common surgical conditions seen in childhood and will be very helpful to the pediatrician and the general practitioner. It is written apparently more for use in this manner than for the teacher or as a textbook.

It gives a very common-sense clinical approach to most of the diagnostic treatment problems involved in the care of these common surgical conditions. The most sympathetic and understanding attitude by the author, who is an outstanding pediatric surgeon, makes it quite evident that he loves children. In discussing controversial subjects—as, for example, undescended testicle—he gives both viewpoints of the controversy before stating his own opinion.

One thing that might be considered lacking would be the references at the end of each chapter, but, as this is not apparently written as an exhaustive textbook type of thesis, the lack of references is understandable.

This book will prove a valuable addition to the libraries of all practitioners who have to deal with children.

JAMES C. OVERALL, M.D.
Nashville

Diseases of Women

FREDERICK W. ROQUES, M.D., JOHN BEATTIE, and JOSEPH WRIGLEY, Editors, 1959. Baltimore: Williams & Wilkins Co. 556 pages. Illustrated. \$8.00.

The tenth edition of "Diseases of Women" by ten teachers is better than ever. The eminently practical, experienced contributors have made it an obstetric-gynecologic handbook for the student. This book should be on the office desk, in the operating room library, and on the bedside table as a handy reference.

The text is divided into 4 parts, namely, anatomy, physiology, inflammatory conditions, and noninflammatory conditions. Data on cervical and vaginal cytology have been added. Cyclic and endocrine interpretations of smears are emphasized. Earlier diagnosis of cancer of the uterus is stressed via cervicovaginal cytology correlated with biopsy and pathologic evaluation. The chapter on vulvitis and vaginitis is concisely organized and informative. The mechanics and treatment of prolapse of the uterus and the important steps in gynecologic operations have been well described.

The beauty of the book is the fact that it is easy to read. It is written in simple, understandable English.

Not infrequently medical students, general practitioners, and obstetric-gynecologic surgeons wish to have access to a book that gives concise viewpoint on a specific topic. Thus they avoid delving through large, detailed tomes. There may be the desire to "brush up" before examinations of a specialty board or for a basic review before operative surgery takes place. This essential guide is useful in bringing out, from a "pigeonhole" of memory, stored information.

WALTER SUSSMAN, M.D.
Philadelphia

Chemistry of Pancreatic Diseases

HARRIS BUSCH, M.D., PH.D., 1959. Springfield, Ill.: Charles C Thomas. 143 pages. Illustrated. \$5.25.

The pancreas is one of the most active exocrine and endocrine glands of the body, demonstrating marked activity of enzymes and hormones and, in this, utilizing a variety of amino acids for protein synthesis. It plays an important part in the synthesis and secretion of metabolically and nutritionally important hormones and enzymes which appear to be under control of both neuro-humoral and humeral mechanisms. The pancreas suffers from diseases which may be related to both excessive and inadequate secretions of its hormones and from disorders which prevent the proper release of its normal secretions. A unique disease of chemical necrosis also involves the pancreas, and there is much which has to be learned about this, both etiologically and therapeutically. The author attempts in this book to assess the present state of knowledge of the biochemical activity of the normal pancreas, to present some characteristics of its internal and external secretion, and to describe some aspects of the chemical pathology of the abnormal states resulting from aberrations of its chemical activity. For the proper therapeutic management of pancreatic dysfunctions, a thorough knowledge of the chemical activity of this organ is required. Dr. Busch has attempted, in a short text, to compile much of the information that is available concerning this structure.

The book is divided essentially into 3 parts: the first deals with the chemistry of the exocrine diseases; the second, with the chemistry of the endocrine diseases; and the third, with the neoplasms of the pancreas.

The biochemistry section presents considerable information about protein synthesis, which appears to be the main industry of this organ, and there is definite consideration given to the amino acids which are taken up by the pancreas; peptide formation; liberation of enzyme precursors; and factors and mechanisms involved in the extrusion of the zymogen granules which have been formed. Considerable attention is devoted to the details of the types of enzymes secreted by the pancreas. These include trypsin, chymotrypsin, carboxypeptidases, amylases, lipolytic enzymes, cholesterol esterase, and pancreatic lipase as well as the depolymerases and hydrolases which attack the phosphoric acid bonds of the nucleic acids, for example, the pancreatic deoxyribonucleates.

The mechanisms of control of secretion of pancreatic juice include a consideration of secretin and pancreaticozymine as well as a consideration of the influence of cholinergic innervation and of administered cholinergic drugs. The author briefly discusses the inhibition of the formation of secretin by the use of carbonic anhydrase inhibitors. Although the enzyme inhibits carbonic anhydrase in the tissues, it does not necessarily follow that the inhibition of secretin formation is directly due to this enzyme inhibition. It may be that this is causing alterations in pH which affect the cells so that secretion is cut down and that it is not a direct effect of the enzyme carbonic anhydrase but an indirect one.

Various tests for plasma pancreatic enzymes are stressed as being of diagnostic importance in dealing with acute pancreatitis, and the biochemical pharmaco-

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BOOK REVIEWS—(Continued)

logic principles essential for this understanding and management of the disease are briefly but cogently presented.

Various aspects of pancreatic insufficiency have been considered, as has hypoinsulinism. The text is finished by a brief discussion of hyperinsulinism and various neoplasms which affect the pancreas.

The text is printed in a clear fashion which facilitates rapid reading. There are some typographic errors which are not important and will probably be corrected in future editions. The book is well documented with references to allow the interested reader to pursue individual subjects to greater detail. This is one of the monographs produced by Charles C Thomas in the series on American Lectures in Living Chemistry and will provide considerable information to the individual who would like to be brought up-to-date quickly on various aspects of pancreatic physiology and pathology. There are 143 pages of concise material. It may not present all of the answers to all questions one might raise concerning pancreatic dysfunction but certainly allows one to be quickly brought up-to-date and to be given the leads for more detailed information if his interest and curiosity desire it.

IRVING H. LEOPOLD, M.D.
Philadelphia

Bone Tumors

LOUIS LICHTENSTEIN, M.D., 1959. *St. Louis: C. V. Mosby Co.* 402 pages. Illustrated. \$12.00.

The first edition of Lichtenstein's "Bone Tumors" was published in 1952. It comprised 310 pages. This second edition has been enlarged to 398 pages. There is added a short chapter on the clinical management of lesions that may be bone tumors and also a foreward from the first edition. Some change in opinion has occurred. In the first edition, the author felt that Ewing's tumor should be treated by radiation alone; he now feels that irradiation plus extirpation of the tumor offers the best hope for the patient. Some chapters have been enlarged, for example, that on multiple myeloma. Many of the author's former opinions remain unchanged in spite of a difference of opinion by other pathologists. Parosteal osteogenic sarcoma, for example, is still not made a separate entity. This is a book which has gained wide recognition in its first edition as an authoritative text on bone tumors. In its second edition, it will continue to be thus acknowledged. It should be in the library of everyone who deals with bone tumors.

JOHN MOE, M.D.
Minneapolis

The Johnson Recording Oscillometer

CARL A. JOHNSON, M.D., 1959. *New York: Pergamon Press.* 112 pages. Illustrated. \$5.00.

This monograph presents a detailed review of a precision-timed, recording oscillometer which provides a means of obtaining a permanent calibrated record of the arterial volume pulse. As such it undoubtedly represents a marked improvement on the usual oscillometer which is neither precise, accurate, nor calibrated. The instrument described herein utilizes a recording droplet of 95 per cent ethyl alcohol to record the arterial volume pulse. It is apparently quite accurate in measuring volume change from the extremities, fingers, toes, temporal artery, intraorbital tissues, and skull. The author begins with a detailed and at times tedious description of

the physical properties and characteristics of the oscillometer. Considerable emphasis is made of the fact that the oscillometer records an arterial volume pulse rather than a pressure pulse. One of the principal uses of the instrument is to obtain blood pressures from various levels of the extremities. It appears that the method of blood pressure recording is somewhat cumbersome in comparison to similar techniques which are available for this purpose, such as plethysmography. In the discussion on arterial blood pressure, unnecessary emphasis is given to obvious and well-known facts in the recording of blood pressure. It is difficult to review this book without making necessary comparisons between the records which are obtained with the recording oscillometer and objective measurements of the same phenomenon which can be made with segmental and digital plethysmographic techniques. The latter studies can be quite as accurate and appear to offer considerably more information than this instrument. The records which are obtained with this instrument show a heavy baseline which is somewhat undesirable. One of the most interesting applications of the recording oscillometer is its use in measuring intra-orbital pressures. Although this has apparently not been explored in detail as yet, it would appear to have an interesting potential. Here again, however, there is unnecessary space given to details of operation of the instrument. The illustrations are of average quality and serve to explain the text, which is at times not too clear. This monograph would appear to have very limited usefulness for the average physician or student in view of its extremely confined content and sphere of interest.

JAMES E. CROCKETT, M.D.
Kansas City, Kansas

The Respiratory Muscles and the Mechanics of Breathing

E. J. MORAN CAMPBELL, M.D., 1958. *Chicago: Year Book Publishers, Inc.* 131 pages. \$4.25.

This extraordinary book by a British author emphasizes the great value of concentrating on a single phase of a subject in order to emphasize and explain its importance. The author has accomplished his objective. In the foreword, Dr. Richard Riley, an American, writes as follows: "The field of respiratory physiology is expanding in all directions, yet so preoccupied have most physiologists been with lung volumes, ventilation, circulation, gas exchange, the mechanics of breathing, the metabolic cost of breathing and the control of breathing that few have paid much attention to the muscles that actually do the breathing. Dr. Campbell has gone a long way toward making up for past deficiencies on this score."

"This reader had not adequately appreciated the need for limitation of voluntary activity of the respiratory muscles. On the basis of electromyographic studies Dr. Campbell demonstrates the existence of automatic restraints in an excellent chapter on the nature of the limitation of maximum inspiratory and expiratory efforts. One then begins to see the place of the muscles of respiration in broader perspective. They are strategically placed not only to perform their essential normal function in breathing but also, if they over-act, to interfere with vital respiratory and circulatory functions. Their freedom of action must therefore be restrained, and the nature of the control mechanisms is of extraordinary interest."

"Until recently little was known about the metabolic cost of breathing and the efficiency of the respiratory muscles, yet Dr. Campbell not only interprets his electro-

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myographic studies in the light of this newer work but presents some important original contributions in this field. He is likewise able to correlate electromyographic findings with pressure-volume changes during the respiratory cycle in a way which is highly satisfactory. In scientific work there is nothing more convincing or more exciting than the convergence of data from independent sources upon a single interpretation."

A fine list of references and an adequate index are included. This is an important book for investigators in respiratory problems, regardless of specialization.

JOHN S. LUNDY, M.D.
Rochester, Minnesota

Fundamentals of Otolaryngology

LAWRENCE R. BOIES, M.D., 1959. Philadelphia: W. B. Saunders Co. 510 pages. Illustrated. \$8.00.

The present edition, like its predecessors, is intended to provide a fundamental knowledge of otolaryngology for the undergraduate medical student and for the general practitioner who does not specialize in otolaryngology but whose patients frequently require examination or treatment for disorders in this field. Dr. Boies and his 7 collaborators have achieved these objectives in a volume which is outstanding for clarity and excellence of organization.

The descriptions of applied anatomy and physiology are supplemented by illustrations of fine quality, particularly in the section on the nose. The inclusion of history forms for the various categories of otolaryngologic disease is a useful feature, and the concise but adequate description of various methods of examination and testing, also aided by ample illustrations, provides a sound understanding of the approach to clinical diagnosis.

Useful office procedures and techniques of minor surgical nature are given clearly and in sufficient detail. Discussion of the major surgical techniques, such as those involving the temporal bone, are confined chiefly to the principles involved, in keeping with the authors' stated purpose. The chapter on medication is up to date and well organized and includes material on methods of home treatment by the patient.

The expanding phases of the specialty of otolaryngology have been recognized by the inclusion of additional material on reconstructive nasal surgery, maxillo-facial injuries, and tumefactions of the neck. The chapters on diseases of the larynx, esophagus, and bronchial tree are necessarily brief but provide adequate coverage for the nonspecialist. For those readers desiring to further pursue a given subject, references are given at the end of each chapter.

CHARLES NORRIS, M.D.
Philadelphia

Microbial Variation

V. D. TIMAKOV, 1959. New York: Pergamon Press. 202 pages. Illustrated. \$6.50.

All biologic phenomena are influenced by both heredity and environment. When the intricate interplay of these is neglected and emphasis is put on one at the exclusion of the other, we have the partisan view. Such is the case with "Microbial Variation," dealing mainly with work carried out at the Gamaleia Institute of Epidemiology and Microbiology in Moscow. As stated by the editor of the volume, "the main significance of the work lies in the experimental confirmation of the basic tenets of Michurin biology."

The bulk of the data deals with microbial variation as influenced by certain changes in the environment. Many of the variations ascribed to induction by the environment may have been due to spontaneous mutation followed by selection, others to sexual phenomena or to genetic transduction (to which no reference is made). Frequently, wholesale transformations of microbial populations are described. It is well known that particular caution should be exercised before claiming environment-induced mutational changes when the change appears to affect the whole population exposed. No such caution is apparent here. Attempts to discriminate critically between mutation and selection on one hand and true induced genetic changes on the other by such established methods as the Luria-Delbrück analysis, Lederberg's replica plating method, or the Cavalli-Lederberg sib selection method are absent.

References in general are few. Treatment of the important world literature on the subject is cavalier. There are only 5 references to the non-Russian literature. The reviewer is thus left with the impression that the book has been written in a scientific vacuum. The little treatise can be recommended to the specialist in microbiology or social psychology as an interesting example of the doctrinaire view in science. The busy physician will not find in it a balanced treatment of an important field.

HERBERT M. HIRSCH, M.D.
Minneapolis

The Physiology of the Newborn Infant

CLEMENT A. SMITH, M.D., 1959. Springfield, Ill.: Charles C Thomas. 497 pages. \$12.50.

The first and second editions of this book have been unexcelled as compilations of valuable physiologic information regarding the fetus and newborn infant. The third edition remains of the same quality. Much of it has been rewritten in an effort to include the ever-increasing research findings of workers in this field. It is, therefore, quite up to date. The author is, himself, an important contributor to the research literature and is thus unusually well qualified to review critically the work of others in the field. Since he is also a clinician, there is more than a little included for everyone with any interest in the newborn. Although it is scholarly enough and contains an adequate bibliography for the basic scientist, it has been written to help the clinician. It is hardly recommended for a quick cover-to-cover session, but, taken in small doses, it may be properly appreciated.

Although each chapter is concluded with a clinical summary, the clinical aspects are by no means confined to these pages. Thorough reviews of such problems of current interest as bilirubin metabolism, the infants of diabetic mothers, and immunology in the newborn period, are excellent. This book, in the reviewer's opinion, is one that is indispensable for any clinician who is responsible in any way for the care of newborn infants. It is highly recommended for inclusion in the reserve and circulating sections of hospital libraries, since its contents are particularly aimed at the physiologic aspects of hospitalized newborn infants. However, the book is not intended to compete with standard works of pediatrics dealing with diseases of the newborn.

A minor criticism is due the crowded arrangement and small type used in the table of normal values on the inside back cover and its adjoining page. Inclusion of values not ordinarily sought as "quick reference" material seems responsible.

ROBERT A. ULSTROM, M.D.
Los Angeles

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BOOK REVIEWS — (Continued)

Hypnosis in Anesthesiology

M. J. MARNIER, M.D., 1959. *Springfield, Ill.: Charles C Thomas*. 150 pages. \$6.75.

This book strips away much of the mystery about the manner in which an anesthesiologist may use hypnosis. The book is very practical. The author makes no intimation that the standard chemical anesthetic agents should be eliminated by hypnosis, although it appears that sometimes such can be done. It would seem that many of the unpleasant pre- and postoperative nervous conditions often encountered can be greatly modified by means of hypnosis. Such modification would mean, in turn, that the work expected of the chemical anesthetic agent would be greatly reduced. Although avoidance of hypnosis in certain cases is not greatly stressed, the following statement should be kept in mind constantly if a novice is to have the temerity to attempt hypnosis without some training in its use and contraindications. The author writes, "These problems will not be encountered by anesthesiologists if care is exercised in the choice of patients so as to avoid hypnosis in individuals with deep psychological disturbances." He explains what hypnosis can and does do. He exercises much commendable caution in describing how hypnosis can be effected and presents many case reports to support his statements. The use of hypnosis in the therapy of pain states is pointed out.

An excellent bibliography on the subject is provided. The book is indexed and printed on good paper and can be easily read. The volume is very much needed, and all physicians who must manage patients subject to nervous stress or states will derive considerable benefit from it.

JOHN S. LUNDY, M.D.
Rochester, Minnesota

Critical Incidents in Psychotherapy

STANLEY W. STANDAHL, PH.D., and RAYMOND J. CORNINI, PH.D., 1959. *New York: Prentice-Hall, Inc.* 396 pages. \$6.95.

As the book jacket suggests, this volume presents 23 super case conferences on psychotherapeutic problems. "Approximately four hundred psychotherapists were invited to submit 'critical incidents' of theoretical, procedural, or ethical interest." Comments and opinions regarding a selected set of these therapy problems were then solicited from a rather large and diverse group of "consultants" representing anthropology, psychology, psychiatry, semantics, and sociology. Finally the editors selected 23 critical incidents that had drawn comment from at least 5 consultants for inclusion in the book. A final chapter, written by the editors, summarizes each of the preceding ones.

The book reads easily and should prove worthwhile reading for anyone engaged in or interested in the problems of helping those in psychologic difficulty through psychotherapy. It is not, nor does it claim to be, a systematic treatment of the theory or practice of psychotherapy. The cases and incidents discussed are not uncommon or atypical, however; similar cases could probably have been drawn from many psychotherapists' files. Likewise, the techniques mentioned are among the more frequently encountered ones.

Being a series of super case conferences, the book shares the virtues and flaws of that institution, both

somewhat magnified by the presence of so many consultants. It stimulates and refreshes by bringing the reader into contact with many different points of view and insights other than his own in regard to the case and incident under consideration. But, as in so many case conferences, one is sometimes left breathless as he watches sweeping psychologic generalizations unfold on the basis of so little factual data. The diversity and lack of congruence between consultants' comments also make it clear that at this point in time there is as yet no correct monolithic approach to psychotherapy; that psychotherapy still remains more art than science.

JOHN O. KANGAS, PH.D.
Minneapolis

Diabetic Manual

ELLIOTT P. JOSLIN, M.D., 1959. *Philadelphia: Lea & Febiger*. 304 pages. Illustrated. \$3.75.

The tenth edition of the "Diabetic Manual" by Dr. Elliott Joslin maintains the same high standards with regard to content and method of presentation as did previous editions. The personal touch abounds throughout, making for easy reading and rapt attention. The more recently employed hypoglycemic agents are conservatively discussed with justified emphasis on the importance of maintaining diet while employing these compounds. Exercise, that often forgotten aspect of management, is frequently mentioned and duly stressed. The discussion of the complications of diabetes is forthright yet understanding and sympathetic. This "Diabetic Manual" continues to deserve the appellation, "The Diabetic's Bible."

G. CLAYTON KYLE, M.D.
Philadelphia

Medical Discoveries — Who and When

J. E. SCHMIDT, M.D., 1959. *Springfield, Ill.: Charles C Thomas*. 555 pages. \$14.75.

Within the limitations of 555 pages, Dr. Schmidt has compiled a dictionary of medical progress from the recorded beginning of man's curiosity about his own body to the present time. Although no work of this type can attempt to be up-to-the-minute in such a rapidly advancing field, the more than 6,000 entries comprise an impressive history in concise, easy-to-read form. Listed in alphabetical order, items related to methods, terms, doctrines, diseases, and operations have been included with complete information as to their founders, dates of discovery, and eponyms and synonyms which might otherwise lead to confusion. A sample entry tells us that the cortical visual center was discovered in 1888 by Salomon Elberhard Henschen, a Swedish pathologist who lived from 1847 to 1930, while information can also be found on such widely varied subjects as the first use of rubber gloves in surgery and the application of lysivane to the treatment of parkinsonism. In addition to subjects directly affiliated with medicine, the reader will find a number of entries dealing with the fields of dentistry, pharmacy, nursing, opticianry, midwifery, chemistry, and physics, all bearing, if only indirectly, upon medical science. The book is recommended to anyone interested in a logically categorized capsule summary of medical history.

NANCY S. HAWKE
Minneapolis

The Journal Lancet

SERVING THE MEDICAL PROFESSION OF MINNESOTA,
NORTH DAKOTA, SOUTH DAKOTA, AND MONTANA

The Specialty of Neurology

Role and Scope of Neurologic Practice in the United States

CHARLES M. POSER, M.D.

Kansas City, Kansas

THE YEARS since the end of World War II have seen a literal revolution in the field of neurology. This revolution consisted primarily in transforming neurology from what had been traditionally a descriptive clinical exercise into a dynamic, therapeutically-oriented specialty. The vision of such leaders as Pearce Bailey, A. B. Baker, Russell DeJong, Francis Forster, Houston Merritt, and Adolph Sabs led during this time to the establishment of the National Institute of Neurological Diseases and Blindness under the auspices of the United States Public Health Service and the foundation of the American Academy of Neurology.

The change in the character of clinical neurology has been both the result of and the impetus for radical advances in the basic neurologic sciences. Thus, neuropathology adopted the techniques of electron microscopy and histochemistry; new biochemical methods and gains made in enzyme chemistry heralded the birth of neurochemistry; the pharmacologists joined forces with the electrophysiologists to give neurophysiology a new direction. Today, a funda-

mental belief in clinical neurology is that diseases of the heart, the liver, and the lungs may result in neurologic signs and symptoms. The study of enzyme systems and metabolic disturbances outside as well as inside the brain is a necessary adjunct to the more formal study of the shape of neurons and the connection of fiber tracts.

One aspect of the growth of the specialty can be gauged by the increase in the number of residencies available for training clinical neurologists. In 1934 and 1935, there were 13 approved programs with a total of 38 residencies. In 1957 and 1958, there were 109 programs offering a total of 398 residencies. Seventy per cent of these residencies were filled.^{1,2}

In terms of the percentage of available residencies filled, neurology stands 22 out of the 26 specialties listed. Table 1 shows the number of residencies available and the percentage that were filled during the last eight years. Since so many demands for trained clinical neurologists and neurologic scientists remain unfilled, the American Academy of Neurology has established a special committee to deal with problems of placement.

During the last fifty years, the face of American medicine has changed, and, with it, neurologic disease has soared in importance. It has been estimated that there are more than 15

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TABLE 1
NUMBER OF APPROVED RESIDENCIES IN NEUROLOGY^{1,2}

Period	Number of approved programs	Total appointments (all years)			Per cent filled
		Offered	Filled	Vacant	
1949-50	69	200	173	27	87
1950-51	80	216	131	85	61
1951-52	78	232	142	90	61
1952-53	81	261	178	86	62
1953-54	76	250	160	90	64
1954-55	79	270	188	82	70
1955-56	96	316	206	110	65
1956-57	103	353	250	103	71
1957-58	109	398	280	118	70

million individuals disabled by neurologic and sensory disorders, almost twice the number of people suffering from heart disease and cancer combined.³

The striking decrease in infant and child mortality has resulted in a sharp increase in the population of mentally retarded and defective children as well as in great numbers of children affected with a variety of pre- or perinatal cerebral defects and injuries, some familial and others not, which usually are erroneously grouped together as "cerebral palsy." In a recent masterly study of the problem of mental deficiency, Masland⁴ pointed out that "Of the 4,200,000 children born annually in the United States, 3 per cent, or 126,000, will never achieve the intellect of a 12-year-old child; 0.3 per cent, or 12,600, will remain below the 7-year intellectual level; and 0.1 per cent will spend their lives as completely helpless imbeciles, unable even to care for their own creature needs."

Probably nothing can bring out this problem more graphically than the fact that each year over 11,000 patients are admitted for the first time to public institutions for mental defectives and epileptics in the United States. The cost of maintaining the patients in such institutions amounted to almost \$103 million in 1951.⁵

Neurologists should keep in mind, in dealing with the rare and curious anomalies and metabolic defects so frequently encountered in the state training schools, Monrad-Krohn's admonition that "the value of old curiosities depends on the light they may throw on general problems."⁶ Too often, institutions for mentally defective children and hopelessly deteriorated epileptic patients have been shunned by the therapeutically minded.

There is a growing number of elderly people and a concomitant increase in the cerebral complications of old age and its diseases. In 1900, 4 per cent of the American population were over the age of 65. In 1950, the figure was 8.1 per cent, and it has been estimated that in 1975, 9.4 per cent will be over 65. Perhaps the increase of our population in the older age groups is even more striking in absolute numbers than in percentages: In 1900, there were 3,080,498 Americans over the age of 65; in 1950, there were 12,271,178—almost a fourfold increase.⁷

From available figures on admissions to hospitals for the care of the mentally ill, it is apparent that organic diseases of the nervous system constitute an increasingly larger percentage of such admissions.

That the diseases affecting the mind in old age are the result of organic deterioration has been

TABLE 2
DISTRIBUTION OF LEADING GROUP OF PSYCHOSES AMONG FIRST ADMISSIONS TO THE NEW YORK CIVIL STATE HOSPITALS⁸

Diagnosis	Year	Percent of admissions	Variation	Rate ^a	Variation
Dementia praecox	1909	19.8	- 33%	25.4	- 55%
	1956	26.1		11.5	
Involuntional	1911	2.5	+ 180%	1.5	+ 360%
	1956	7.0		6.9	
Manic depressive	1909	7.7	- 73%	4.5	- 60%
	1956	1.9		1.8	
[Senile	1909	11.6	+ 40%	6.8	+ 130%
	1956	16.2		15.8	
[Cerebral arteriosclerosis	1912	2.9	+ 725%	1.8	+ 1200%
	1956	23.9		23.3	
Alcoholic	1909	10.8	- 11%	6.2	
	1956	6.4		6.3	
[General paresis	1911	13.3	- 94%	8.2	- 90%
	1956	0.8		0.8	

^aRate per 100,000 population

⁸"Neurologic" disorders accounted for 40.9 per cent of all first admissions.

TABLE 3
FIRST ADMISSIONS TO ALL TYPES OF PSYCHIATRIC
FACILITIES FOR MENTAL DISEASE
IN THE UNITED STATES IN 1951^a

1. ALL PATIENTS	262,281
2. PSYCHOSES	180,475
3. General paresis	3,291†
4. Other forms of central nervous system lues	1,008†
5. With epidemic encephalitis	248†
6. With other infectious diseases	606†
7. Alcoholism	14,789
8. Due to drugs or exogenous toxins	1,620
9. Traumatic	1,019†
10. With cerebral arteriosclerosis	23,555†
11. With other disturbances of circulation	1,350†
12. With convulsive disorder	2,554†
13. Senile	21,681†
14. Involutional	14,427
15. Due to other metabolic diseases	1,472†
16. Due to new growth	492†
17. Others with organic central nervous system changes	3,136†
18. Manic depressive	14,274
19. Schizophrenia	54,500
20. Paranoia and paranoid conditions	5,985
21. Psychopathic personality	1,799
22. With mental deficiency	3,103†
23. Other and undiagnosed psychoses	8,566
24. PSYCHONEUROSES	24,560
25. WITHOUT PSYCHOSES	52,163
26. Epilepsy	2,206†
27. Mental deficiency	2,405†
28. Alcoholism	30,794
29. Drug addictions	3,487
30. Personality disorder due to encephalitis	128
31. Psychopathic personality	5,090
32. Primary behavior disorder	2,337
33. Others and unclassified	5,716
34. MENTAL DISORDER UNDIAGNOSED	3,275
35. NO MENTAL DISORDER FOUND	3,208

^aIncluding state, county, city, and private hospitals for mental disease, psychopathic hospitals and psychiatric sections of general hospitals.

†Indicates what has been considered "neurologic"

known for many years. How prominent these causes of mental disease have become in recent years is reflected by the 1,200 per cent increase in the rate of cerebral arteriosclerosis as a cause for admission to the New York state hospitals during the past forty-nine years (table 2). Together, arteriosclerosis and senile psychoses constitute over 40 per cent of admissions to the New York state hospitals during the year 1956.⁸

The figures for the year 1951 for the first admissions to all types of psychiatric facilities from the entire United States are equally revealing (table 3). Almost 30 per cent of these admissions are on account of diseases that may be considered neurologic.⁵ Alcoholism was left out of what we have considered "neurologic" diseases, although a great many alcoholic patients undoubtedly exhibit signs and symptoms of organic neurologic involvement. It is idle to argue whether

a patient with a Korsakoff's psychosis or a Wernicke's encephalopathy belongs to the psychiatric or the neurologic service.

If the present trend continues—and there is no reason to doubt that it will—the neurologic scientist's interest in these organic mental diseases must increase. The science of neurology began in the "insane asylums" almost one hundred years ago. Now neurologists must return to the asylums, this time armed with promising new investigative and therapeutic tools.

The health of the nation will become more and more dependent upon the available number of well-trained clinical neurologists and upon the advances and discoveries of basic neurologic science research. At the present time, how well is the need for neurologic talent being filled?

One way of evaluating this is on a geographic basis. According to figures published in April 1958 concerning physicians certified in neurology by the American Board of Psychiatry and Neurology, 11 states have not a single certified neurologist. There are still 6 states in which there is no specialist certified in both psychiatry and neurology.⁹⁻¹¹

In order to make a conservative estimate of the availability of neurologic service in each state, it is arbitrarily assumed that one-third of the physicians certified in both neurology and psychiatry devote at least some of their time to the practice of neurology. In most areas, this estimate may be high; in others, it is probably low. It is also undeniably true that some physicians certified in psychiatry only, as well as a large number of neurosurgeons and many internists and pediatricians, also provide neurologic care. There can, however, be no doubt that certification in neurology is the only way to be sure that the quality of such care is high.

For each state and territory, a "corrected total" has been found by combining the number of specialists certified only in neurology with one-third the number of specialists certified in both neurology and psychiatry. In order to point up the relative paucity or density of neurologists in each state, the number of physicians certified in neurosurgery and those certified in dermatology are compared with those in neurology in table 4. The choice of dermatology was indicated by the fact that, in many areas and in many medical schools, dermatology, like neurology, is considered a subspecialty of internal medicine and practiced by internists.

Table 4 shows that, in most areas, there are many more dermatologists than neurologists. In the great metropolitan areas of California, the District of Columbia, Illinois, Massachusetts,

TABLE 4

<i>State</i>	<i>N</i>	<i>NP</i>	<i>T</i>	<i>Dens.</i>	<i>Rank</i>	<i>NS</i>	<i>Dens.</i>	<i>Derm.</i>	<i>Dens.</i>
Alabama	3	3	1	0.13	40	6	0.19	12	0.39
Alaska	0	0	0	0.00	17	0	0.00	0	0.00
Arizona	0	2	1	0.11	36	1	0.57	9	1.28
Arkansas	1	3	2	0.10	42	4	0.21	5	0.26
California	43	110	75	0.71	6	71	0.67	200	1.88
Canal Zone	0	0	0	0.00	17	0	0.00	1	2.00
Colorado	5	10	8	0.61	9	6	0.46	15	1.15
Connecticut	7	27	16	0.80	5	20	1.00	29	1.45
Delaware	0	1	2	0.66	8	1	0.33	3	1.00
District of Columbia	12	26	21	2.66	1	18	2.25	27	3.39
Florida	2	11	6	0.27	24	13	0.46	27	0.97
Georgia	3	9	6	0.17	33	12	0.35	15	0.44
Hawaii	0	2	1	0.20	30	3	0.00	5	1.00
Idaho	0	1	1	0.17	33	1	0.17	2	0.34
Illinois	11	59	31	0.36	14	24	0.27	82	0.94
Indiana	5	10	8	0.20	30	13	0.33	22	0.56
Iowa	3	8	6	0.23	29	8	0.31	14	0.54
Kansas	4	6	6	0.31	19	5	0.26	10	0.52
Kentucky	2	5	4	0.14	36	8	0.27	7	0.24
Louisiana	4	11	8	0.30	21	9	0.33	15	0.55
Maine	2	1	3	0.33	15	1	0.41	2	0.22
Maryland	10	17	16	0.70	7	10	0.43	22	0.96
Massachusetts	21	74	19	1.01	3	29	0.62	45	0.96
Michigan	15	16	20	0.31	19	21	0.33	49	0.76
Minnesota	28	29	38	1.26	2	16	0.53	22	0.73
Mississippi	0	0	0	0.00	47	2	0.09	3	0.14
Missouri	1	21	11	0.28	23	14	0.36	36	0.92
Montana	0	0	0	0.00	47	2	0.33	2	0.33
Nebraska	1	4	2	0.15	35	5	0.38	10	0.76
Nevada	0	0	0	0.00	47	2	1.00	3	1.50
New Hampshire	1	3	2	0.10	12	3	0.60	5	1.00
New Jersey	10	21	18	0.32	18	8	0.16	61	1.27
New Mexico	0	2	1	0.11	36	1	0.11	1	0.57
New York	61	261	119	1.00	1	68	0.16	317	2.14
North Carolina	7	9	10	0.21	27	12	0.29	27	0.66
North Dakota	0	0	0	0.00	17	0	0.00	2	0.33
Ohio	12	32	23	0.29	22	36	0.15	80	1.01
Oklahoma	1	1	2	0.09	11	5	0.23	13	0.59
Oregon	6	5	8	0.53	10	6	0.40	16	1.06
Pennsylvania	16	68	39	0.37	13	27	0.26	118	1.12
Puerto Rico	1	0	1	0.04	16	1	0.18	2	0.09
Rhode Island	3	4	1	0.50	11	3	0.37	5	0.63
South Carolina	1	1	2	0.10	12	3	0.11	11	0.52
South Dakota	0	0	0	0.00	17	0	0.00	2	0.28
Tennessee	2	7	1	0.12	41	19	0.58	18	0.54
Texas	11	27	20	0.26	25	31	0.10	74	0.96
Utah	1	0	1	0.11	36	4	0.57	7	1.00
Vermont	1	0	1	0.25	26	2	0.50	3	0.75
Virginia	2	12	6	0.18	32	12	0.36	24	0.72
Washington	5	8	8	0.33	15	9	0.37	17	0.71
West Virginia	0	3	1	0.05	45	5	0.25	6	0.30
Wisconsin	4	12	8	0.21	27	9	0.26	23	0.68
Wyoming	0	1	1	0.33	15	0	0.00	0	0.00

1. N: Number of physicians certified in neurology by the American Board of Psychiatry and Neurology, (through March 1958 examinations).
2. NP: Number of physicians certified in both neurology and psychiatry by the American Board of Psychiatry and Neurology.
3. T: Total number of physicians assumed to practice neurology. This number is arrived at by adding one third the number of physicians certified in both neurology and psychiatry to the number of physicians certified in neurology alone.
4. Dens.: Number of physicians practicing neurology per 100,000 inhabitants (1950 census). Dens. = T divided by population.
5. Rank: Ranking of states according to density.
6. NS: Number of physicians certified in neurologic surgery by the American Board of Neurological Surgery (through June 1956).
7. Derm.: Number of physicians certified in dermatology by the American Board of Dermatology (through June 1956).

New York, Pennsylvania, and Minnesota neurologists outnumber neurosurgeons, while the latter are more numerous in the more rural states.

In states with relatively small populations, there is too often a disproportionately small number of neurologists available. Undoubtedly, one of the main reasons for this is that most specialists prefer to be located near large medical centers for teaching and research. This, of course, does not obviate the fact that a great part of the population has little chance to receive first-rate neurologic care. That the trend of the neurologic specialist is still primarily toward big-city practice is evident from the fact that, during the last two years, 30 of the 56 certifications in neurology were given to physicians practicing in the areas previously mentioned. An encouraging fact is that the newly certified neurologists were distributed over 22 states and that Puerto Rico obtained its first neurologist.

It is generally assumed that a neurologist should take care of a population of between 75,000 and 100,000 inhabitants. If the higher figure is accepted, only the District of Columbia, Minnesota, Massachusetts, and New York are adequately covered.

Examination of the status of undergraduate training in neurology in the different states is revealing. The great neurologic training centers are in New York, the District of Columbia, Boston, Minneapolis, and Ann Arbor. This is why these areas are well supplied with neurologists, and it is also a tribute to the training programs in effect there. The best source of residents for any training program is its own medical school. Since the basic source of supply is the undergraduate program, a great deal of attention must be paid to it. This is especially important in the large state-supported institutions, since the specialists trained in state institutions are those most likely to settle within the states themselves.

The following information was obtained from the most recently available catalogs of the different medical schools. In 21 schools, there is a separate, autonomous Department of Neurology. Of these, only 7 are in state-supported institutions. Neurology forms a section of the Department of Medicine in 33 schools and exists jointly with psychiatry in 18 schools and with neurosurgery in 2. In 6 schools, there is no special Section of Neurology.

In 24 medical schools, no courses in neurology are listed beyond neuroanatomy and neurophysiology courses in the preclinical years. In many instances, it is difficult to tell from information available in the catalogs how many of the

courses listed in neurology are required or elective and what percentage of the graduating classes receive formal or bedside clinical instructions in neurology.

It is probably also of interest that the 13 states which have no specialist certified in neurology are, with the exception of Mississippi, also states without a four-year medical school—Alaska, Arizona, Delaware, Hawaii, Idaho, Montana, Nevada, New Mexico, North Dakota, South Dakota, West Virginia, and Wyoming.

The problem of undergraduate neurologic education has had earnest consideration by the leaders of American neurology. A symposium on Neurological Education was held in conjunction with the June 1953 meeting of the American Neurological Association.¹²

At that conference, Dr. A. B. Baker stated:

"In my opinion, this problem of interest in post-graduate training in neurology can be resolved and the answer obtained by two processes. First is a program of public education so that the people of our country realize the scope of the neurologic problem and the illnesses that fall into this field and make demands upon us for such medical skills and medical knowledge, thus creating a real demand for the neurological specialty. Second, a more adequate program of undergraduate teaching of neurology in all medical schools so that every graduating student has an adequate concept of the broad, dynamic aspects of neurology. Through such undergraduate teaching, I feel certain that enough interest can be created in the medical student so that a large number will feel inclined to enter the field of this specialty."

Four years later, in 1957, Dr. Baker pointed out that, "Of some 7,500 physicians graduated each year from our medical schools, less than 50 per cent received an organized course in neurology given by a neurologist so that they might be able to recognize neurologic illnesses or apply the known treatment procedures in this field . . ."

SUMMARY AND CONCLUSIONS

Advances in the general medical care of the population of the United States have resulted in an ever increasing number of pediatric and geriatric patients. The prevention and care of mental deficiency on the one hand and arteriosclerosis and senility on the other are areas of medicine of growing importance. Neurologists should recognize and shoulder their responsibility in those fields. Greater emphasis is also needed for a more equitable geographic distribution of trained clinical neurologists. One means of achieving this goal is to improve the undergraduate neurologic curriculum and to stress the importance of proper neurologic care for our steadily rising and longer living population.

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THE COMMON COMMUNICABLE diseases of childhood appear to be responsible for a greater number of central nervous system disorders than is generally supposed. For every patient with clinically obvious encephalitis, there are hundreds more who have slowed electroencephalographic activity during the acute or postacute period of measles, mumps, chickenpox, scarlet fever, or rubella. Fever is not responsible for these electroencephalographic abnormalities, since normal tracings have been obtained in patients having rectal temperatures over 104° F.

Electroencephalographic tracings of 1,298 children with contagious diseases were studied in relation to clinical evidence of encephalitis. The results are as follows:

Diagnosis	Total number of patients	Symptoms of encephalitis (abnormal EEG)	No symptoms of encephalitis (abnormal EEG)
Measles	717	37 (37)	680 (344)
Mumps	126	16 (16)	110 (34)
Chickenpox	253	16 (16)	237 (53)
Scarlet Fever	156	— —	156 (33)
Rubella	46	1 (1)	45 (6)

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The Present Status of Contact Lenses

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CONTACT LENSES are certainly not new and have now become an integral part of our everyday life. Wearing them is no longer a curiosity or a novelty but a routine prescription by the practicing ophthalmologist or optometrist.

The early corneal lenses were first introduced by E. Kalt in 1888, Zeiss in 1912, and E. Rakos in 1938. These lenses were made of glass and had many shortcomings. Even in 1930, we used a trial set of 15 to 30 glass lenses of varied corneal and scleral curves. The fitting was done by trial and error, and the lenses were ordered from Germany. A buffered solution was used in the lenses and had to be replaced periodically. The wearing time was very limited, and there were frequent clouding and visual disturbances requiring the removal of the lenses for half an hour to an hour for a period of rest.

In the late 1930's, plastic lenses were introduced. This material made it far easier to work with the lenses, as the shape could be altered by grinding or buffing for minor corrections. There was also a safety factor, as the lenses could not be broken by dropping them or by a direct blow to the eye. To begin with, we used a molding material and took a cast of the anterior segment of the eye. The correction required for vision was then ground into the corneal part of the lens, and this was fitted to the patient. These lenses were of very large size, covering the anterior surface of the eye almost from corner to corner. In the late 1940's, the smaller lens, or corneal lens, was introduced. This kindled a new interest in the lenses and increased their usefulness.

DESCRIPTION

The optical principle of the contact lens is to replace the refractive power of the cornea with a curved lens of known dioptic power. This is accomplished by placing the cornea between

two fluids of the same refractive index, thereby destroying the refractive power of the cornea. This is similar to the lack of distortion that is seen through a curved piece of glass when it is submerged in water. Human tears and the aqueous humor of the eye have essentially the same refractive index, and the refractive power of the cornea is thereby destroyed.

There are two main types of lenses: (1) scleral, or fenestrated lenses, and (2) corneal lenses, including the micro lens. The scleral lens, as the name implies, extends partly over the white portion of the eye on either side of the cornea. Such lenses are approximately 23 mm. in diameter and about .7 mm. thick. There is a small hole at the periphery of the corneal portion which allows for an interchange of the fluid on either side of the lens. This also allows an escape of carbon dioxide, which is given off from the front surface of the cornea. This is necessary to prolong the wearing time. In the case of the fenestrated lens, fluid is placed in the lens before it is inserted in the eye. It is held in place by adhesion between the scleral portion of the lens and the scleral part of the eye, there being minimum clearance of the corneal portion. Some corneal contact is allowed and expected, especially with movement of the eye.

The corneal lens is by far the most popular lens and the most versatile in its usage. These lenses are small, approximately 8 to 11 mm. in diameter and approximately .18 to .62 mm. thick. They are ground on three radial curves to facilitate fitting. On observation, very little difference can be seen in the curvature of the lens, as it has the appearance of a continuous curve. The corneal lens is fitted over the anterior surface of the cornea and held in place by adhesion. This is similar to two flat pieces of wet glass which are very difficult to pull apart, but one can be moved over the surface of the other very readily. As the contact lens moves over the anterior surface of the cornea, carbon dioxide is allowed to escape from the exposed surface.

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USES

Who wears contact lenses? It is estimated in the 1959 contact lens survey that approximately 6,000,000 people are wearing them. This figure seems relatively high, but it is very evident that the number of wearers is increasing steadily. About 65 per cent of the wearers are women. In the 14 to 25 year age group, 60 per cent of the wearers are women. Vanity was the reason given for usage in 79 per cent of the women, other reasons being vision, athletics, and social acceptance. Of the men, 39 per cent wore the lenses for masculine ego. The remainder chose them for convenience, athletics, and occupation.

Of the specific uses for contact lenses, keratoconus is probably the most spectacular example. In these cases, the cornea is cone-shaped rather than evenly curved. Ordinary spectacle lenses are of very limited to no help. Here the replacement of the cornea by the contact lens is the solution to improved visual acuity. In many of these cases, vision can be improved from 5/200 or 10/200 to essentially normal vision. Needless to say, these people tolerate their lenses far better than the others because they are so essential to their everyday life. Recently, corneal lenses have been used in these cases with a fair degree of success. This means contact of the cornea with the posterior surface of the lens at the apex. This has proved to be of some help in retarding the progression of the keratoconus. If the apex is very high, a corneal lens cannot be used, as it cannot be held in place. A fluid lens is then used, and in these cases the apical cone can be cleared with the contact lens. If the patient is in the presbyopic age, ordinary spectacles must be worn to give him additional help in his reading.

In cases of irregular corneal scars, such as burns or lacerations, the astigmatism cannot be corrected with ordinary glasses. Here again, the optical replacement of the cornea by the contact lenses has proved of value. Corneal or fenestrated lenses are used, depending on the defect.

In the field of athletics, contact lenses are now almost standard equipment for many sports. Certainly, in football and swimming, contact lenses are the only visual aids of any practical value. In basketball, hockey, and such sports, ordinary glasses can be worn without too much difficulty, but they are not visually as good as a contact lens. In football and swimming, the larger lens, or fenestrated lens, is the one of choice. This is because it will stay in place much better and will stand the abuse of the rougher sports. It is possible to use the smaller lenses, but one must always keep his eyes partially squinted or

the lens may wash or flip out. There is no apparent danger in using contact lenses in rough sports, as they are well protected by the lid and the close fit of the lens over the surface of the eye serves as a splint.

In unilateral aphakia, contact lenses have become a real boon. The average individual who has had a cataract removed from one eye and has good usable vision in the other will not tolerate the high correction of a cataract lens in the former and a normal correction in the latter. With the cataract lens, the retinal image is greatly enlarged over that in the other eye, and the disparity causes the average patient too much discomfort. However, when a contact lens is worn over the operated eye, the retinal images are of approximately the same size and can be fused without discomfort or difficulty. In the older individual, this is probably not practical, as he does not care to bother with contact lenses. However, in the young adult, who has cataracts secondary to trauma, this is of definite value. An eye that is not used, such as a cataractous eye, has a tendency to turn out and to be a definite cosmetic blemish. Removal of the cataract can be done with a high degree of success, and a lens can then be fitted to the operated eye three or four months after surgery. This will be tolerated quite well in the usual case as the patient once again obtains binocular vision which is of great advantage to him. The eyes will then usually realign if there has been some deviation. However, if the cataract has been present for a long period of time, strabismus surgery may be indicated in addition to the cataract surgery. In bilateral aphakia, which is also rather common in the adult between 40 and 50 years of age, contact lenses are being used more and more each day. The advantage of using them in this case is again cosmetic, but they also help visual efficiency. It is well known that the field of vision is quite limited in a patient with cataract lenses, and, when the eyes are rotated, there is a definite prismatic effect from the lenses which is quite disturbing to the patient. In these cases, contact lenses are fitted to be used for everyday wear. Bifocals are then prescribed, using a plain lens above and a reading lens which is required in the lower segment. The patient can then carry on activities and use bifocals or reading glasses whenever close work need be done.

In the cosmetic field, myopia is by far the most common reason for wearing the contact lens. As mentioned, they are worn most commonly by women for this defect, but more and more men are also using these lenses. It is very evident that people in public life, such as actors, ac-

tresses, and lecturers, find these lenses to be of extreme value in their ordinary activities. In some occupations, especially in outside work, they have a distinct advantage. With the contact lens, the sailor or the structure worker who has difficulty with ordinary glasses due to steaming and wetting during rain or adverse weather conditions only needs to blink his eyes and his field of vision is clear again.

PROCEDURE

How are contact lenses fitted? A routine procedure in the fitting of contact lenses is here tabulated. In larger towns, we have the services of contact lens technicians who are associated with the various optical companies. They are able to do most of the technical fitting of the lens and will carry out most of the steps here enumerated. If the practitioner is in a smaller town, he will have to do these things himself after being properly instructed in the fitting of the lenses.

If the patient is interested in contact lenses, "negative pressure" is used. We point out the shortcomings of the lenses—the limited usage, intolerance, and expense—and stress the relative simplicity of ordinary glasses. However, if the patient is still definitely interested in buying contact lenses and has a legitimate reason for using them, we do a complete refraction. His vision is improved to the best possible acuity and the correction recorded. This is sent to the technician with a reading of the vertex distance if the correction is more than 5 diopters of either myopia or hyperopia.

The patient is then shown what lenses look like, how they are worn, and what he can expect from them. A trial lens is then put in place in either one or both eyes. He is asked to sit in a chair and look down. It is emphasized to the patient that there is no correction in these lenses and that his vision will not be improved with them. However, he will have the same sensation with these lenses that he will when he receives lenses of his own. These lenses are allowed to remain in place for ten or fifteen minutes, and usually the patient starts to move his eye before that time is up. He may find that when he moves his eye up, there is definite discomfort, and, as a result, he prefers to keep looking down in the lower field. He is also advised that he will be aware that the lenses are in his eyes and be semiconscious of them for about a month. He is then shown how easy it is to remove the lenses by looking down and pulling his lower lid temporally.

The cost of a pair of lenses is then discussed.

In this area, I believe the average charge for corneal lenses is \$150. If the patient decides to go ahead with the fitting, financial arrangements are made at that time, usually requiring a deposit of one half the total price of the lenses. A keratometer reading, which measures the anterior curvature of the cornea in the vertical and horizontal meridian, is then taken. The doctor's prescription for the refraction is used, using only the spherical portion of the correction. A chart is used to transpose these figures into the size of the lens required. The patient's lens is then either made up or taken from stock.

Three appointments are made for the future fittings. During the first one, the patient is checked for tolerance and wearing ability. This usually lasts approximately one hour. On the next visit, he is taught how to insert and remove the lens and is started on a wearing schedule. It has been found that if a patient has the ability to take the lenses in and out himself, he has more interest in them and adjusts to them at a faster rate. He is advised to wear the lenses for one hour three times a day for the first two days. Each wearing period is then increased by one-half hour for the next two days. It is emphasized that the wearing period should always be a comfortable one. If, at the end of a half-hour or an hour or whatever length of time it might be, the lenses become uncomfortable, he is advised to remove them promptly. However, he is encouraged to use them as much as possible, especially during the first month.

After about two weeks, the patient returns to the doctor, who checks the fitting of the lenses and makes sure that the correction is adequate. It is emphasized that the patient should wear the lenses each and every day. If he wears them only occasionally, his tolerance will not be good and he will tend not to use them steadily. If, at the end of the month, it is found that the patient will not tolerate lenses and he has put forth an honest effort to do so, he is advised that he is not a good candidate for contact lenses. It is then a general policy that the deposit already made will be forfeited but he will not be held responsible for the remainder of the contracted price. This time period is a very flexible factor, and usually doctors continue to work for two or three months with any patient who has made a continued attempt to wear the lenses.

There are many other facets to the contact lens problem. One of the newer innovations is the use of bifocals in contact lenses. These are really in an experimental stage and are not widely prescribed. In these cases, the lens is slightly wider than the ordinary corneal lens and has a rim

of approximately 3 mm., which is a stronger correction than the central area. The use of this lens requires a very cooperative patient, as he must look through the center of the lens for all distance vision. When he looks down, the lens rides up on the cornea, and he looks through the stronger edge of the lens, which is in the pupillary area. As is evident, the field would be quite limited and one would have to hold his eyes in a rather fixed position to obtain the vision he so desired. There are at least four different types of bifocal lenses, but none of them have proved very satisfactory to date.

Another type of lens is the colored lens for use either as a sunglass or to change the color of one's eyes. This has no practical importance for the average person, but it is used occasionally on the stage and in movies.

DISCUSSION AND SUMMARY

We feel that contact lenses are now practical

and can be used by almost anyone with a moderate amount of discretion. Optically, they are ideal; practically, they leave something to be desired. There have been no known cases of any permanent damage from the average use of contact lenses, and we inform the patient that they should be worn with ordinary care. There has been some notoriety given to the possibility of contact lenses preventing myopia, but this is not substantiated. It has been found that myopia is not as markedly progressive with the use of contact lenses as with ordinary glasses, and they may possibly have some effect on the cornea.

Contact lenses are now a common prescription by the refractionist. They have their limitations but certainly offer many advantages, especially in the designated cases. Corneal lenses are the most popular and most easily worn. Although it is doubtful whether contact lenses will replace regular spectacles, we can be sure of their extended usage.

XEROPHTHALMIA INVOLVING ATROPHY of the lacrimal gland and accessory tissue eventually leads to total blindness. The parotid gland offers a convenient source of permanent lubrication, and transplantation of the duct is not difficult.

The operative site is infiltrated with 1 per cent Xylocaine containing 1:1,000,000 Adrenalin to induce local ischemia in the periductal connective tissue. A No. 3 ureteral catheter is then passed through the duct papilla and up to the parotid gland, causing the duct to bow outward. A 2-cm. vertical incision is made over the anterior borders of the masseter muscle, crossing the course of the duct. An imaginary line from the ear lobule to a point midway between the nasal ala and the upper lip vermilion bisects the incision line. In order to preserve the facial nerve branches, blunt dissection is done parallel to the course of the duct, and the duct is completely mobilized from gland to papilla. With the duct papilla centered vertically, a cuff of oral mucous membrane 2 by 3 cm. is incised. With excess fat and connective tissue removed, the cuff is formed into a tube to extend the duct. After closure of the wound, a small Penrose drain is inserted. A subcutaneous tunnel is formed from the origin of the parotid duct to a point one-third of the way from the lateral end of the conjunctival cul-de-sac by blunt dissection. The duct and attached cuff are drawn through the tunnel, and the margins of the cuff are attached to the conjunctiva of the inferior fornix with interrupted sutures.

After closure of the skin incision, Neosporin ophthalmic solution is placed in the conjunctival sac and the eye is dressed. Two days following surgery, the eye pads and pressure dressing and the drain in the oral wound are removed.

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The Diagnosis of Cardiac Arrhythmias

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THE PROBLEM of differential diagnosis of an abnormality in cardiac rate or regularity is not an unusual one for the physician engaged in a general medical practice. The most common of these abnormalities will be described and the electrocardiographic features briefly outlined. For ease in presentation, the arrhythmias have been grouped according to their clinical characteristics.

RAPID "REGULAR" RHYTHMS

The most common tachycardia is sinus tachycardia. It consists of an accelerated discharge of the normal cardiac pacemaker (sinoatrial node) to a rate above 100 per minute and usually not exceeding 150 per minute. The rhythm is quite regular, but the rate may vary somewhat on repeated counts and may slow with expiration and become faster with inspiration (sinus arrhythmia). The rate will also quicken with exercise. Carotid sinus or eyeball pressure produces a gradual slowing of rate followed by a gradual quickening of rate after release of pressure. The electrocardiograph shows the normal sequence of P-QRS conduction (figure 1a). Sinus tachycardia may be present in a number of clinical conditions, among which are fever, anemia, hyperthyroidism, and anxiety.

Paroxysmal tachycardias are supraventricular (atrial or nodal) or ventricular in origin. The onset is abrupt, and the patient usually recognizes the sudden onset of palpitation and tachycardia. Termination of the attack is equally abrupt. Atrial and, to a lesser extent, nodal tachycardias are seen quite frequently. They are apt to occur in healthy individuals, with a history of the initial attack occurring under the

age of 40 years. The rhythm is extremely regular; the rate does not vary with exertion and is usually between 150 and 250 beats per minute. Carotid sinus or eyeball pressure will have no effect on the rate and rhythm or will terminate the tachycardia suddenly. Carotid sinus pressure is more successful in nodal tachycardia. The electrocardiographic feature of atrial tachycardia is a rapid, regular rhythm with a normal P-QRS relationship (figure 1b). The P waves, however, will be of unusual contour. This will be particularly appreciated if a previous electrocardiograph on the same individual is available for inspection. If the rate is very rapid, the P wave may be superimposed on the preceding T wave and be very difficult to identify. The electrocardiograph in nodal tachycardia resembles that seen in atrial tachycardia except for the configuration and position of the P wave (figure 1c). Since atrial stimulation occurs in a retrograde fashion from the atrioventricular node in nodal tachycardia, the P wave will be negative, especially in standard leads 2 and 3. The P wave will be immediately in front of the QRS (PR less than 12 seconds), hidden in the QRS itself and not identifiable, or immediately following the QRS. The position of the P wave depends on the site of the pacemaker within the atrioventricular node and the degree of resistance to retrograde conduction. At times, nodal tachycardia may be differentiated clinically from atrial tachycardia if large jugular venous pulses, or cannon waves, are noted. These occur when the right atrium contracts against a closed tricuspid valve.

Ventricular tachycardia is much less common than the supraventricular tachycardias and usually occurs in individuals with organic heart disease. It tends to be less regular than atrial or nodal tachycardia and does not respond to carotid sinus pressure. Infrequently, it may be

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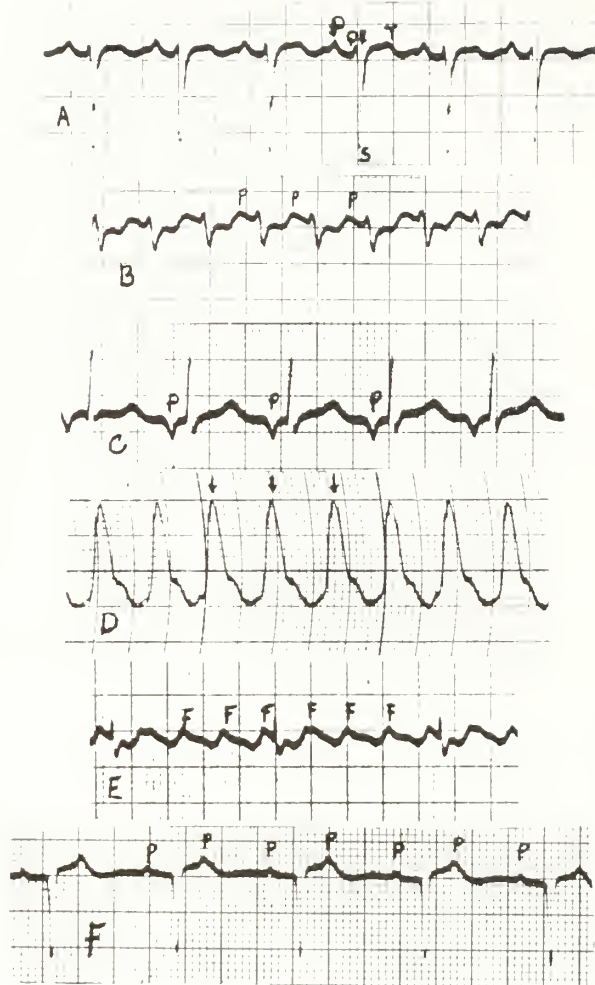


Fig. 1

recognized clinically if occasional large jugular venous pulses are seen or if the first heart sound varies in loudness. These two findings are a result of the independent activity of the atria and the ventricles in this disorder. The electrocardiograph shows a regular or slightly irregular rhythm with widened, notched QRS complexes (figure 1d). The T wave is usually opposite in direction to the main QRS deflection. When P waves can be made out, it will be noted that they are occurring at a different rate than the QRS complexes and that no consistent P-QRS relationship exists. The atria and the ventricles are contracting independently. When P waves cannot be identified, it may be difficult to differentiate supraventricular tachycardia with bundle-branch block from ventricular tachycardia. In these instances, it can be helpful to review a pretachycardia electrocardiograph on the patient to see whether or not a bundle-

branch block pattern was present at that time and to compare the QRS configuration with those present during the tachycardia.

Atrial flutter is characterized by a rapid atrial rate, usually 260 to 340 per minute, with the ventricles responding only to every other atrial beat or, at times, to every third or fourth atrial impulse. It occurs predominantly in people with heart disease. It may occur paroxysmally but usually tends to be more chronic in duration. Carotid sinus pressure may cause an abrupt slowing of the ventricular rate by increasing the degree of block present. Thus, an atrial rate of 300 may result in a ventricular rate of 150—2:1 block—which may be slowed abruptly to 75—4:1 block—by carotid sinus pressure. The electrocardiograph shows the rapid F waves occurring regularly and tending to blend one into the next, producing a “sawtooth” appearance. These waves are usually negative in standard leads 2 and 3. The QRS complexes occur regularly after every other F wave or, in higher degrees of block, after every third or fourth F wave (figure 1e). At this point, paroxysmal atrial tachycardia with block should be mentioned. The electrocardiograph shows a rapid regular sequence of P waves with ventricular responses (QRS) only after every other P wave—2:1 block—(figure 1f) or, less frequently, after every third or fourth P wave. The P waves are quite sharply formed and are separated from one another by a stretch of level baseline in contrast to the F waves of atrial flutter which tend to merge one into the following. Atrial tachy-

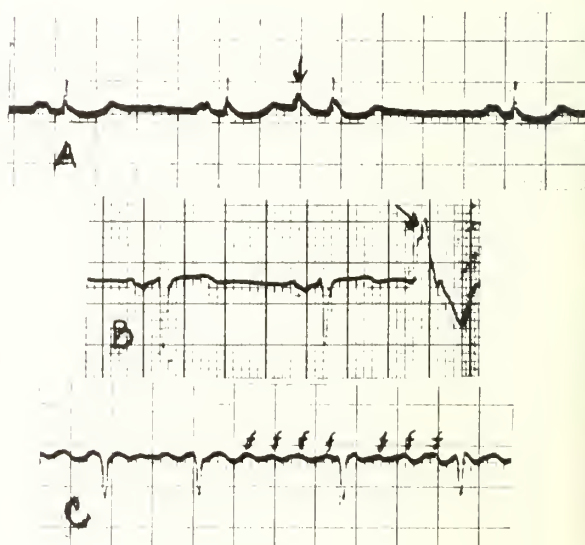


Fig. 2

cardia with block is being reported more frequently recently, secondary to digitalis intoxication precipitated by the use of diuretic agents producing potassium loss.

IRREGULAR RHYTHM

The most common cause of irregularity in cardiac rhythm is premature beats. These may arise from a focus in the atria, atrioventricular node, or the ventricles. They are recognized on auscultation as an interruption of the normal cardiac rhythm by a premature beat followed by a compensatory pause and subsequent resumption of regular rhythm. They may occur only occasionally or so frequently as to follow every normal beat. The electrocardiograph pictures this interruption in rhythm. Atrial premature beats are distinguished by a P wave of different shape or magnitude than the preceding P waves (figure 2a). The QRS complex usually shows no change; occasionally, it will be somewhat slurred and widened if portions of the ventricles are still refractory due to the nearness of the preceding beat. If the atrial premature impulse is extremely early, the ventricular response may be blocked entirely. Nodal premature beats are distinguished by the position of the P wave immediately prior to or after the QRS complex or by the lack of an identifiable P wave. This is due to retrograde stimulation of the atria from the atrioventricular node as previously described for nodal tachycardia. The QRS complex of ventricular premature beats is widened, notched, or slurred, and the T wave is usually opposite in direction to the main QRS deflection (figure 2b). At times, a P wave may be seen in the terminal portion of the QRS or the S-T interval as a result of retrograde conduction and stimulation of the atria. The distance between the QRS prior to the ventricular premature beat and that following it will be found to be twice the distance between QRS complexes elsewhere (full compensatory pause). In the case of atrial premature beats, this measurement is somewhat less than twice the usual cycle length.

Atrial fibrillation is also a commonly seen form of cardiac irregularity and occurs usually in the presence of heart disease. The atrial impulses reach the rate of 350 and more per minute and the atria themselves do not actually contract but remain distended in diastole and show fibrillary twitching. The atrial impulses are manifested on the electrocardiograph as rapid, irregular waves of low amplitude and variable shape and are called "f waves." Only a portion of the atrial impulses succeed in traversing the normal conduction pathways to produce a ven-

tricular response. The ventricular rate is therefore slower, usually 140 to 150 per minute, and totally irregular. On auscultation, variation in loudness of the first heart sound as well as the total irregularity of rhythm will be noted. Determinations of the pulse rate taken at the wrist will be found to be 10 or more beats per minute less than the cardiac rate determined at the apex with the stethoscope (pulse deficit). This finding is a result of the extreme irregularity of rhythm.

Ventricular contractions occur at varying stages of ventricular filling. Those contractions, which take place when only small amounts of blood are present in the ventricles, will not produce a pulse at the wrist, and the pulse deficit results. Digitalis decreases the number of atrial impulses transmitted through the conduction pathways to the ventricles and thus slows the ventricular rate in this arrhythmia. The electrocardiographic findings consist of an absence of P waves, the presence of "f waves," and the total irregularity of rhythm of the QRS complexes (figure 2c).

HEART BLOCK

This section is concerned with rhythm disturbances which are a result of varying degrees of failure of the atrial impulse to be transmitted through the normal pathways and produce a ventricular response.

A rather rare variety of this type of disorder is complete blocking of the atrial pacemaker im-

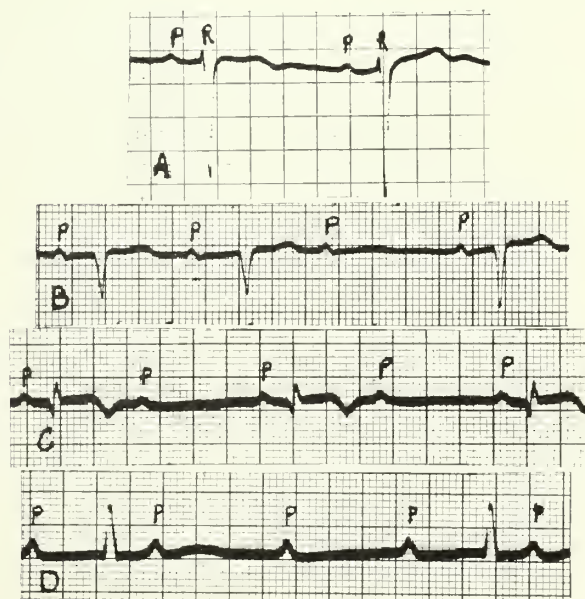


Fig. 3

pulse—sinoatrial block—so that neither atria nor ventricles are stimulated to contraction. This is manifested by a complete dropping out of one cardiac cycle, and auscultation will reveal a pause in the cardiac rhythm which will be twice the duration of the pause between the preceding beats. There will be no evidence of atrial activity during the pause as indicated by the absence of a jugular pulse wave. The electrocardiograph shows interruption of a sequence of normal beats by a period of inactivity equal to two normal cycle lengths. Very rarely, the atrial pacemaker may be blocked on two or more successive occasions and the pause, or period of inactivity, will then endure for corresponding multiples of the normal cycle length.

A more common variety of block is that in which the impulse is conducted normally through the atria but is delayed in its passage through the atrioventricular junctional tissue. The elapsed time between atrial activity and ventricular activity is thus increased. The electrocardiograph in this situation shows an increase in the P-R interval to beyond .20 seconds (figure 3a). This is termed first degree heart block. No abnormality in rate or rhythm is detectable on auscultation but decreased intensity of the first heart sound may be noted.

Further increase in degree of delay of the atrial impulse results in second degree heart block. The Wenckebach phenomenon may result. This consists of a progressive increase in the P-R interval until finally one of the P waves fails to initiate a ventricular response. The succeeding P wave starts the cycle over again. Auscultation reveals a regular rhythm with a gradual and slight increase in rate, then a sudden pause slightly less in duration than twice the preceding cycle, and then resumption of the regular rhythm (figure 3b). With a slightly more pronounced degree of block, failure of ventricular stimulation may occur more frequently and regularly producing pauses every third beat—3:2 block—and thus a coupled rhythm, or every other beat—2:1 block. If the jugular pulse is

observed, it will be noted that the regular jugular pulse due to atrial contraction continues regularly through the pauses. The electrocardiograph shows regular P waves with lack of a following QRS complex every second, third, or fourth time depending upon the degree of block—2:1 block—(figure 3c).

The next, and highest, degree of block is that in which all of the atrial impulses are consistently prevented from reaching the ventricles—third degree, or complete, heart block. As a result, the ventricles contract in response to a pacemaker located in the lower portion of the atrioventricular node or the bundle of His. Since the inherent rate of this pacemaker is slower than the normal atrial pacemaker, the heart rate and pulse usually range from 30 to 50 per minute. Further slowing in rate or temporary failure of this pacemaker will result in giddiness or Adams-Stokes syncope. On auscultation, the striking finding is the slow, regular cardiac rhythm. The first heart sound will be noted to vary in intensity and clearness due to the occasional superimposition of atrial contraction upon ventricular contraction. At times, the faint, regular sound of atrial activity may be heard continuing independently of the slow regular ventricular contractions. The jugular pulse will be noted to continue regularly and at a faster rate than ventricular contractions as heard with the stethoscope. The electrocardiograph demonstrates the complete independence of atrial and ventricular rhythm. The P waves and QRS complexes occur regularly, but each at its own rate. The QRS complexes may appear normal or may be widened, notched, or slurred, depending upon the location of the ventricular pacemaker (figure 3d).

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A POOR PROGNOSIS may be expected when nucleated red cells are found in the peripheral blood of patients with congestive heart failure. Although the cells may disappear temporarily, death frequently occurs within a short time. Premature or abnormal release of nucleated red cells may be caused by bone marrow anoxia.

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The Diagnostic Uses of Radioactive Materials in Cardiovascular Diseases

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RADIOACTIVE ISOTOPES are now considered necessary in all types of medical research. There are few published articles dealing with investigative procedures in which they are not mentioned as important tools. In more recent years, these investigations have been assuming a practical role in clinical medicine as diagnostic procedures. This is particularly true in the field of cardiovascular pathology.

I have been asked to discuss the latter category in a general manner and shall refrain from using technical descriptions and mathematical calculations. The basis for this report is a review of the ever-increasing current literature and my own personal experience in dealing with radioactive material which extends back to 1946.

Early in the "atomic age," blood flow was studied by using the tagged red cell and following its course through the circulatory system. The red cell was labeled with either radioactive phosphorus or chromium; this method is still in use and has been proved to be a valuable procedure. Human serum albumin labeled with radioactive iodine (I^{131}) is now used extensively in almost all circulatory studies. The substance is prepared by associating the human serum albumin with I^{131} . The isotope combines with the albumin without changing the properties of the protein molecule. The labeled agent has the same characteristics as human albumin. This substance is designated as IHSA.

If a proper amount of IHSA is injected intravenously, it acts as a plasma diluent mixing with the patient's plasma and behaves in the same physiologic manner. It is thus a biologic tracer substance which emits gamma radiation.

Blood volume of an individual can be determined by removal of a sample of his blood fifteen to twenty minutes after IHSA has been given intravenously. The known activity of the injected IHSA is then compared with the activity of the blood sample, and by utilizing the

hematocrit value in calculation with the two readings of activity, we arrive at the actual blood volume. The entire process can be accomplished in approximately forty-five minutes. This relatively short time is extremely important when the information is needed quickly.

Cardiac output can be determined by measuring the concentration of IHSA as it passes through the heart. If the degree of concentration of the labeled agent is recorded for a definite period of time, the volume of flow per unit of time may then be determined. The equipment necessary for this determination will not be found in the usual hospital laboratory. Thus, the practical value of such a procedure is somewhat limited in the scope of clinical medicine.

Circulation time, or the velocity of blood flow between two points, is important information when one is dealing with diseases of the peripheral vascular system. It is possible to obtain this information by using IHSA as in other circulatory studies. This is of particular value in determining results of sympathectomy.

In our laboratory, we have used this substance as an agent in the diagnosis of congenital peripheral communications, particularly in the presence of small shunts. The IHSA is injected rapidly into the femoral artery under local anesthesia. A shielded detector located over the right atrium picks up the radioactive effect when IHSA arrives at this point. The time element is recorded by means of a graphic recorder. If arteriovenous shunts are present in the peripheral vascular tree, the time of flow is definitely less than the normal values.

Radiocardiography is a good term. It is a method of measuring graphically the flow of radioactive blood through the cardiac chambers. The originators of this procedure have been able to construct a normal curve and gross deviations are interpreted to indicate certain phases of cardiac pathology. The test should have clinical value in detecting ventricular dilation. Some authors state that it is possible to distinguish between ventricular hyperplasia and ventricular

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dilation and between uncomplicated emphysema and cor pulmonale with dilation of the right side of the heart. It is my impression that these investigators are correct in their assumptions and that, with further refinement of sensitive detectors, radiocardiography will be a very important clinical aid in the diagnosis of heart disorders. It is now possible to detect a left-to-right shunt, as found in some congenital abnormalities of the heart, without the use of intravascular catheterization.

Other substances are being used in studies of circulation through the heart. Radioactive sodium was the original tracer to be used and it is still useful in many instances. A new substance, Renografin (radioactive ^{131}I labeled sodium and methylglucamine diatrizoate), is used for these studies also. It has a shorter biologic half-life than I ^{131}I SA, that is, it is retained in the body for a much shorter period.

Newer methods are being developed each year. It is now possible to detect right-to-left intracardiac shunts without femoral artery puncture. Recently radiocardiography has been carried out by using an inhalant as the tracer substance. Radioactive methyl iodide is inhaled into the lung and then makes its way to the left side of the heart, after passing through the pulmonary capillary system and pulmonary veins. The hemodynamics of the left side of the heart can be studied extensively. This method obviates the use of the cardiac catheter. After further refinement of equipment has been accomplished, this procedure will increase in value as an aid in diagnosing congenital heart defects.

All of the methods of radiocardiography are being improved with time, and it is not at all unrealistic to believe that in the not-too-distant future cardiac catheterization will be "a thing of the past."

Quantitative determination of body fluids is important in heart disease. I shall not discuss the procedures used for this beyond stating that

radioactive hydrogen, deuterium, and tritium are the substances used.

I have discussed chiefly the use of radioactive materials in the study of cardiovascular diseases. Only the more practical procedures have been mentioned. There are many more that are in stages of development and refinement. In another five years, the field will have expanded markedly. Most of this expansion will come from the development of better and more sensitive equipment. We may also anticipate that more efficient tracer substances will result from continuing research.

CONCLUSIONS

1. Radioactive methods of cardiovascular investigation in physiologic studies and in cardiac disease have proved to be practical.
2. Newer and better tracer substances may be expected as research in this field continues.
3. It can be predicted that, in the near future, cardiac catheterization will be outmoded in studying congenital cardiac defects.

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THE INCIDENCE OF hyperuricemia in patients with myocardial infarction is similar to that of associated hypercholesterolemia. Of 50 patients with acute myocardial infarction, serum uric acid values were more than 6 mg. per cent in 26 and more than 5 mg. per cent in 42. Serum cholesterol concentration was greater than 250 mg. per cent in 43. Determination of serum uric acid value may be an additional means of assessing predisposition to coronary heart disease.

P. M. KIDN and G. B. PROZAN: Hyperuricemia—relationship to hypercholesterolemia and acute myocardial infarction. *J.A.M.A.* 170:1909-1912, 1959.

Limitations of Electrocardiography

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ELECTROCARDIOGRAPHY has become a necessary adjuvant in the evaluation of the cardiac patient. This is still a laboratory test, and proper judgment should be given to an electrocardiographic interpretation in its application to the patient. By definition, electrocardiography is the science which deals with the study of the electrical forces produced by the heart muscle during the cardiac contraction and relaxation cycle. The electrocardiogram has definite limitations but is useful in the following conditions:

1. Coronary artery disease
 - a. Myocardial infarction—serial tracings are imperative.
 - b. Coronary insufficiency
2. Ventricular hypertrophy
3. Myocarditis
 - a. Deficiency states
 - b. Rheumatic fever
 - c. Diphtheria
 - d. Toxic effects from infections
 - e. Pericarditis
4. Control of medications
 - a. Digitalis
 - b. Quinidine
5. Wolff-Parkinson-White Syndrome—the electrocardiogram is diagnostic.
6. Nonspecific but highly suggestive diagnostic changes may be found in:
 - a. Pericarditis with effusion
 - b. Constrictive pericarditis
 - c. Beriberi
 - d. Obesity
 - e. Hypokalemia
 - f. Hyperkalemia
 - g. Hypocalcemia
7. Pulmonary embolism
8. Cardiac arrhythmias—the electrocardiogram is diagnostic.
9. Auricular hypertrophy

The electrocardiogram does not tell us the state of the heart valves, coronary arteries, cardiac reserve, or the presence or absence of congestive heart failure. Conditions such as bundle branch block, inverted T waves, or evidence of hypertrophy do not indicate a serious prognosis. These changes must be interpreted in the light of

clinical findings. A brief appraisal of some of the electrocardiographic changes are:

Electrocardiographic positions of the heart. In most instances, these are not significant clinically, but a horizontal or semihorizontal type of heart may indicate left ventricular enlargement. A vertical or semivertical type of heart may correlate with right ventricular enlargement.

Axis deviations. The electrocardiographic interpretation of left axis deviation can correlate at times with left ventricular enlargement due to either hypertrophy or dilation. Left axis deviation alone is usually accepted as a normal finding.

Right axis deviation may occur normally in infants but, in adults, may correlate with right ventricular enlargement or, in thin individuals, with a vertical type heart.

There is, as a rule, no quantitative relationship between the electrocardiographic diagnoses of axis deviation and cardiac enlargement.

Ventricular hypertrophy and/or strain patterns. Left ventricular strain pattern is a pattern which has a left axis deviation plus ST and T changes, particularly in the standard leads. This correlates, as a rule, with left ventricular enlargement due either to hypertrophy or dilation. This is an electrocardiographic diagnosis.

Right ventricular strain is an electrocardiographic diagnosis which tends to correlate with right ventricular enlargement secondary to hypertrophy or dilation. This follows chronic lung diseases or severe mitral stenosis or occurs with acute pulmonary infarctions.

Intraventricular block group. Intraventricular block results from a delay in the passage of the excitatory process within the conduction pathways below the bifurcation of the bundle of His. The common forms of intraventricular block are complete right bundle-branch block or complete left bundle-branch block. There are also others, of which the most common is the diffuse intraventricular block. In the diffuse type, the only electrocardiographic finding is a prolonged intraventricular conduction time.

The common causes are: (1) coronary arteriosclerosis, (2) myocarditis, (3) fibrosis of the

myocardium, (4) calcification of the heart, (5) gummata, (6) tumors, (7) congenital defects, and (8) drugs—digitalis and quinidine.

The electrocardiographic interpretation cannot be correlated with the clinical diagnosis. In younger people, a bundle-branch block may be found without any evidence of heart disease. An intermittent or suddenly appearing intraventricular block is of importance in verifying an underlying myocardial disease.

Myocardial infarction. The most important use of the electrocardiogram is in the diagnosis of myocardial infarction. Electrocardiography is the most precise method for detecting this condition and for differentiating it from others which may display a similar clinical symptomatology. From the onset, and for a variable time interval following this, most cases of myocardial infarction produce a distinctive series of alterations of the ventricular complex. These changes take place over a period of days, weeks, or months. Residuals can often be detected many years after an acute episode. Nearly all infarctions of the myocardium are due to coronary heart disease. The electrocardiographic changes may not occur for several days after the clinical onset of a myocardial infarction. The patient may have all the clinical characteristics of acute coronary insufficiency or of an infarction but may have a normal or nearly normal electrocardiogram. The changes, even though minor, may persist for a short or a longer period of time. There is no true correlation between these electrocardiographic changes and the clinical course of the patient with a myocardial infarction.

Angina pectoris (coronary insufficiency). The electrocardiogram is often normal. Shortly after the seizure, the electrocardiogram may show nonspecific T and ST changes. These findings, correlated with the history, may establish the diagnosis. The electrocardiographic changes are reversible within minutes or in an hour.

The hypoxic test or standard exercise test, such as the Master's tolerance test, may cause electrocardiographic changes that will substantiate the diagnosis of coronary insufficiency. The

hypoxic test is dangerous and not commonly used, and the Master's tolerance test may be negative despite classic anginal pain. The best exercise test is to have the patient do the same activity which produces the pain and, when pain occurs, take tracings at four- to five-minute intervals for twenty minutes. These usually show positive changes in many patients.

Pericarditis. The electrocardiogram depicts the changes of the underlying myocarditis and has characteristic changes which, with serial tracings, give rather distinctive changes during the acute and healing stages. Pericarditis with effusion may not give a typical picture.

Arrhythmias and heart block. The electrocardiogram is absolutely diagnostic in the differentiation of the arrhythmias and in heart blocks.

Drugs. Digitalis and quinidine may produce nonspecific electrocardiographic changes.

Electrolytes. The electrolyte disturbances, such as produced by altered potassium levels in the sera, can produce electrocardiographic changes which are nonquantitative. These do not correspond with serum potassium levels, and, at times, a normal electrocardiogram can be obtained in hyperkalemia when a high serum sodium is present.

CONCLUSION

There are many limitations in electrocardiographic interpretations as related to clinical diagnosis and treatment. The electrocardiogram is still a laboratory test and proper judgment should be given in its correlation with the clinical picture. The value of the electrocardiogram is proportional to the ability of the clinician to apply the interpretations as related to the clinical course of the patient.

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ACUTE BENIGN PERICARDITIS may result from infection with Coxsackie virus. Observation of complement-fixing or neutralizing antibody titers and isolation of virus from stools or pericardial fluid will confirm diagnosis. Symptoms may be prolonged, with friction rub sometimes persisting for as long as ten weeks.

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Staphylococcal Pseudomembranous Enterocolitis

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PSEUDOMEMBRANOUS ENTEROCOLITIS is a non-specific, pathologic diagnosis that is actually quite ancient. Credit is generally given to J. M. T. Finney¹ for the first case report in which this descriptive pathologic term was used. He reported the case of a young Negro who underwent gastroenterostomy for a cicatrizing ulcer of the pylorus. Postoperative complications set in, however, and the patient expired. Postmortem examination showed that which was felt to be a classical pseudomembranous enterocolitis. This case was reported in 1893.

Case reports on this subject since have been rare until medical science entered the antibiotic era. Interest in this pathologic diagnosis has been renewed because of the recent recognition of the clinical and bacteriologic diagnosis of a specific form of pseudomembranous enterocolitis due to staphylococcal infection of the bowel. Recognition of this specific etiology for this ancient syndrome has caused a complete reexamination of not only this new infection but the whole spectrum of pseudomembranous enterocolitis.

PATHOLOGY

The pathologic process is nonspecific and may be essentially identical to that produced by heavy metal poisoning, uremia, various septicemias, and bacillary dysentery.² Whether the pathology is due to direct invasion by the staphylococcus or to continuous production of enterotoxin is not known. Other factors are undoubtedly involved also. The condition may well be similar to staphylococcal food poisoning. However, in that condition, the enterotoxin is preformed before being ingested. In food poisoning, it is this enterotoxin that actually causes the disease and not the effects of the staphylococci themselves within the bowel. Sargalla and Dack³ studied the enterotoxin-producing qualities of 33 strains of micrococci isolated from

cases of enteritis after antibiotic therapy. They found that 30 of the strains produced the enterotoxin.

Any area of the bowel from the esophagus to the rectum may be involved. However, the most common areas are the ileum and the colon. In a series of 94 cases reported at the Mayo Clinic,⁴ involvement was noted most frequently in the small intestine (44 cases), the small and large intestines (24 cases), and the large intestine alone (12 cases). Grossly, the bowel is dilated and contains much fluid. Up to 5,000 cc. has been drained with a trocar. The serosa is reddened, and the mucosa shows a yellowish to brownish pseudomembrane formation. This membrane is friable and may occur in plaques or involve entire segments of the bowel for several feet. The membrane is easily removed, leaving the mucosal surface often diffusely injected but many times appearing quite normal. This pseudomembrane can be missed very easily if the bowel is washed out with water. That is why specific attention must be paid to ascertain its presence. The usual practice of having a morgue attendant open the bowel and wash it out immediately after a cursory inspection has undoubtedly been the cause of many missed diagnoses of pseudomembranous enterocolitis.⁴

Penner and Bernheim,⁵ in 1939, reviewed 40 cases of this syndrome and presented what has remained a rather classic pathologic description. They noted marked distention of the capillaries and venules first in the submucosa and then in the mucosa. This was followed by submucosal edema and focal hemorrhages. Next was noted focal necrosis of the tips of the mucosal folds. This was followed by a spreading and fusing of the necrotic foci. In advanced stages, the necrosis was accompanied by an inflammatory cellular reaction. Hale and Cosgriff² described the pseudomembrane as necrotic mucosal debris with inflammatory cells and clumps of bacteria. They noted that occasionally the bowel was thin, but perforation was extremely rare. In general, then, the process is confined to

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the mucosa and the submucosa with deeper extension being very rare.

INCIDENCE

Statistics on the pathologic diagnosis of pseudomembranous enterocolitis are few and far between and difficult to evaluate. Likewise, in the evaluation of the staphylococcal portion of this syndrome, it is difficult to quote the statistics. Previously, the diagnosis was one usually based on a definite pathologic picture found at post-mortem.¹ Subsequently, cases were included on clinical grounds. These were based on the occurrence of a certain group of symptoms with or without bacteriologic proof of the overgrowth of the staphylococcus in the intestinal contents. Most case series are a veritable hodgepodge of cases based on some or all of the criteria previously listed. Some series include surgical and nonsurgical cases also. Therefore, it is not felt at this time that large scale statistics are available.

The Mayo Clinic staff has written extensively on this problem over a period of many years. Many of their conclusions have laid the foundations for current concepts in this condition. Pettet and associates⁴ report a series of 94 cases. These were all based on autopsy diagnoses after surgery. The twenty-eight-year period from 1925 to 1952, inclusive, is a basis for their study. This conveniently divides itself into a fourteen-year period initially when no antibiotics were used and a subsequent fourteen-year period, which may be called the antibiotic era. The most striking fact about their study was that the incidence of cases in the first fourteen years was 45, and the incidence in the second period was 49. In other words, there was no statistically significant increase in cases between the preantibiotic and the antibiotic eras. They concluded that antibiotics could not be shown to have influenced an increase in this syndrome. However, many other authors report that their impression is that this is not correct. Unfortunately, no one else has a series anywhere near as large to draw upon to refute this Mayo Clinic statement.

Due to the increased interest in diagnosing this syndrome, its record for antemortem diagnosis improved markedly. There were no correct diagnoses before death in any patient before 1948. Since 1948, the Mayo Clinic group noted a correct diagnosis in 14 of 26 cases before death. Unfortunately, of 65 records that they felt were adequate for clinical study, only 9 cases had stool cultures before death. Only 1 of these grew out of staphylococci.

Dixon and Weismann⁶ reported one of the

earlier series of cases in 1948. They included 23 cases in their study. In these relatively early days, they played down the role of infection, since they did not find the usual organisms of bacillary dysentery and the parasites on routine stool cultures. In those days, little or no search was done for staphylococcus. One interesting statement made in their study is that the condition seemed to be increasing in incidence "in spite of preoperative sulfonamides." Today, we might say that the condition was increasing in incidence *because of* increasing use of preoperative antibiotics. Kramer,⁷ in 1948, also reported a case with bacteriologic proof of staphylococcal enteritis following streptomycin therapy by mouth. This was certainly one of the earliest cases reported with proof by bacteriology. Speare⁸ reported a collection of 22 cases from the literature in which death occurred in 19. He also reported 8 cases of his own. Of these, 6 were postoperative and 2 were nonoperative cases. Staphylococci were isolated from 5.

In contrast to this, Lepley and Smith⁹ reported 16 cases of staphylococcal enterocolitis, none of which were fatal. They also reviewed the literature and collected 66 cases. They found that 73 per cent of the patients had staphylococci in the stool. The fatality rate was 54 per cent. Weismann and Twitchell¹⁰ reported 16 cases with 8 deaths. However, 5 of their cases were mild and unproved either by pathology or bacteriology.

Terplan and associates¹¹ reported 8 fatal cases. All patients had been extensively treated by antibiotics, either pre- or postoperatively. Prohaska and co-workers¹² reported a series of 7 cases in which there were 4 successive deaths and then 3 dramatic survivals after treatment with ACTH.

Another of the many reports from the Mayo Clinic¹³ studied 14 nonsurgical cases in the eleven-year period from 1940 to 1950. In 7 of these cases, the enterocolitis was thought to be the direct cause of death. However, in 7, the pathologic changes were not thought to be quite extensive enough to account for death. Markley and associates¹⁴ reported a recent review from September 1958. Their series consists of 14 patients, of whom 7 had surgery. Unfortunately, only 2 of the patients had stool cultures.

About the only conclusion that can be drawn from the foregoing series of figures is that the pathologic syndrome of pseudomembranous enterocolitis is fairly rare but carries a very high mortality, probably considerably above 50 per cent. This is true whether it is due to a specific etiology, such as staphylococcal bowel infection, or to other known nonspecific causes.

ETIOLOGY

As stated previously, a widely diverse group of agents can cause pathologic changes virtually identical to those being described in the bowel. The intestine is limited in the number of pathologic responses it may make.⁴ Most recent writers on this subject choose to divide the causes into the antibiotic and the idiopathic. The association with surgery has been extensively explored.

A striking correlation with carcinoma of the colon has been noted at the Mayo Clinic.⁴ Of the series of 94 cases of pseudomembranous enterocolitis that occurred postoperatively, 41 were associated with carcinoma of the colon. Peptic ulcer was associated in 11 cases, cholecystitis in 9, and carcinoma of the stomach in 7. The rest of the cases were scattered over a miscellaneous group, including several types of surgery outside the abdomen.

The incidence of this syndrome associated with carcinoma of the colon was so striking that 10 per cent of the operative deaths with carcinoma of the colon at the Mayo Clinic were due to this particular postoperative complication.

Penner and Bernheim⁵ in one of the earlier papers in 1939, incriminated shock as the prime cause of this syndrome. They felt that this was the basic factor common to all their 40 cases. Most authors since then, however, have tended to feel that the shock is a result and not a cause of the pathologic process.

There has been great difficulty reproducing this syndrome in animals. Establishing a staphylococcal infection in order to do this has defied most investigators so far. However, one group has reproduced that which it feels is a very similar syndrome by transfusing incompatible human blood to dogs.¹⁵ This usually produces a pseudomembrane in the jejunum and ileum. There is also a hemorrhagic necrosis of the superficial mucosa and a thrombosis of capillaries in the mucosa of the intestinal tract. These authors feel that the syndrome is caused by "intravascular clotting in the mucosal capillaries of the gastrointestinal tract during a period of so-called compensated shock." They feel that this compensated shock is invariably produced by any surgical procedure of magnitude. Recalling that the closest association in human beings is with carcinoma of the colon, they offer this as a reason for the increased incidence of thrombosis. Therefore, a human being may have an episode of intravascular coagulation accompanied by surgical trauma, which causes thrombi to localize in his intestinal mucosal capillaries.

They concluded that these 2 conditions are common both to their experiments and to the production of human pseudomembranous enterocolitis.

Fourteen cases collected at the Mayo Clinic which were not operated upon were found to have associated pathologic conditions in 3 major areas: (1) obstruction of the large intestine, (2) cardiac disease, and (3) infection. All of these patients died in shock.¹³ Markley and associates,¹⁴ in a series of 14 cases, noted a striking common denominator of impairment of the blood supply to the gastrointestinal tract. This was due to such things as volvulus, superior mesenteric thrombosis, adhesions, and strangulated hernia. They actually noted occurrence of all the usual factors—previous antibiotic treatment, staphylococcal infection, intestinal obstruction, shock, and predisposing surgical procedures. However, they felt that the main common denominator to be gleaned was the impaired blood supply to the intestine. The response of the intestine to this stress was the production of pseudomembranous enterocolitis. Weismann and Twitchell¹⁰ agree with the multiple causations of this syndrome.

Prior to the last few years, a thorough search for staphylococci in the bowel contents was not done. It was not until the late 1940's and early 1950's that serious complications of staphylococcal overgrowth from the broad-spectrum antibiotics began to be realized. In one of these earlier studies on therapy in pneumonia, Jackson and associates¹⁶ reported a series of 91 cases. Superinfection with staphylococcus in the lungs or bowel was their greatest problem and was felt to be the direct cause of death in at least 4 cases. A Johns Hopkins group reported an interesting sidelight on this problem with the diagnosis of an outbreak of staphylococcal enterocolitis in chinchillas.¹⁷ Animals being raised on a commercial farm had been fed Aureomycin pellets regularly on the vague basis that this was a "nutritional antibiotic supplement." They developed a syndrome similar to that of human beings with an overgrowth of staphylococci in the bowel. Treatment consisted of stopping the pellets and giving the animals a four-day course of bacitracin and neomycin in their drinking water. Recovery from the syndrome was noted, and it has not recurred since the Aureomycin pellets were discontinued.

It is of interest to follow the writing of Prohaska¹² on this subject. In 1954, he reported a series of 7 cases in which this syndrome occurred postoperatively. He had 4 consecutive deaths and 3 subsequent dramatic survivals with

ACTH. He noted definite factors against the staphylococcus being the etiology of the syndrome in his cases, since 3 of his patients did not have any antibiotics preoperatively, they got better instead of worse with ACTH, and stool cultures showed staphylococci on only one occasion. Also, he pointed out that the disease was known well before the advent of antibiotics. However, his later paper, written in 1956, is designed to establish 4 points:

1. Pseudomembranous enterocolitis is an infectious disease produced by micrococcus pyogenes.

2. Staphylococcus doesn't establish itself in the normal flora of the gastrointestinal tract.

3. The shock and fatal course of these patients are enhanced by the continual production of enterotoxin.

4. The effect of the enterotoxin may be negated by the use of ACTH and may even be lifesaving.

He cites his results in 3 cases with spectacular recoveries and dramatic reversal of clinical picture within twenty-four hours after treatment with ACTH. As noted, he feels that the action of ACTH is against the enterotoxin.

Other causes of this syndrome have been cited on various occasions but generally are not well accepted. Poth,¹⁹ who has written extensively on intestinal antisepsis, does not feel that the diarrhea from broad-spectrum antibiotics is due to yeasts because yeast formation is even greater after neomycin has been given and diarrhea did not occur after the use of this drug. The incidence of candida albicans following broad-spectrum antibiotic therapy has been studied, but the incrimination of this agent as a pathogen is extremely difficult to establish, since it is so ubiquitous in health and disease.²⁰ Vitamin deficiency has also been cited as a possible factor.²¹ A rare syndrome entitled "enteritis necroticans" and apparently due to infection with *Clostridium welchii* has some features in common but apparently is not generally related to this particular family of diseases.²²

CLINICAL FEATURES

The chain of events leading to this condition probably includes most of the following factors:⁹

1. A carrier of resistant staphylococci is present.

2. A normal bowel flora is depressed by pre- and postoperative antibiotics.

3. The staphylococcus manufactures its enterotoxin.

4. Staphylococcus invades the wall and produces mucosal necrosis and a massive fluid ex-

travasation and severe diarrhea.

5. The fluid and electrolyte losses cause significant hypovolemia and shock.

The main prerequisites of staphylococcal enteritis, then, are bowel pathology, antibiotic suppression of the normal bowel flora, overgrowth of the staphylococcus, and cross-infection as a source of the pathogen. The patient involved is classically a postsurgical patient, especially one who has had a large bowel carcinoma. The other main group susceptible to this condition is, of course, that group which has had prolonged broad-spectrum antibiotic therapy. Being confined in a hospital helps immensely in picking up a resistant strain of staphylococcus. Cairns⁹ is quoted as showing an increase from 25 to 68 per cent in resistant strains of staphylococcus in the nose and throat in patients hospitalized from one to eight days. Generally, figures quote less than 25 per cent of non-hospitalized personnel as having resistant staphylococci harbored in their noses and throats. The figures for hospital personnel and patients are probably at least in the neighborhood of 50 to 75 per cent.⁸

Johnston and associates²³ subdivide this particular disease into 3 main clinical groups:

1. The choleraic type, which is characterized by an explosive, profuse, watery diarrhea; nausea and vomiting; abdominal cramps; distention; fever; and shock.

2. The ileus type, which is characterized by increasing abdominal distention with copious amounts of fluid obtained by suction. Usually the patient does not have diarrhea. Nausea and vomiting occur, and shock is usually not as dramatic as it may be in the first group.

3. The precipitous shock type, in which mild gastrointestinal symptoms soon proceed into shock, with hypotension and the usual accompaniments of a rapid, thready pulse; pallor; and sweating.

Diagnosis is difficult, especially in the latter 2 types postoperatively. Other authors have also agreed with the descriptive terms of the severe diarrhea which may occur. This has been characterized as choleraformic, with "rice water" stools.⁹ Diarrhea may well be one of the more serious symptoms in this syndrome, and its presence makes the diagnosis easier in many cases. The incidence of diarrhea is variously reported as anywhere from 30 to 100 per cent.² The important thing to realize is that it is by no means strictly necessary to be compatible with the clinical syndrome.

Turnbull²⁴ emphasizes the diagnosis of postoperative staphylococcal enteritis on clinical

grounds. Often, the patient is too critically ill to wait for such things as bacteriologic cultures. Attention is called to the fact that the syndrome usually has a rather abrupt onset, two to seven days postoperatively. Five stages are listed:

1. Abdominal discomfort or pain followed by diminished bowel sounds and then distention.

2. A rise in the pulse to 120 per minute or more. The temperature also starts to rise, and the urine output begins to drop.

3. The most important symptom at present is the diarrhea or emesis of huge amounts of fluids.

4. The blood pressure drops, and shock supervenes.

5. The serum protein and hemoglobin may drop.

Turnbull also uses the term "rice water stools" and adds another, "Micrococcic cholera." He states that he has been disappointed with the results of gram stain smears obtained by rectal swab. Many times he has found that these were negative with cultures later overgrown with staphylococcus. However, even the presence of a negative smear and culture does not rule out the staphylococcus as the etiologic agent. Terplan¹¹ found positive cultures at postmortem but not in the stool during the life of 2 of his 8 fatal cases. In any event, certainly one of the first things to do is make a rectal swab smear with gram stain and culture on blood agar. The white blood count is often surprisingly normal, although the patient is critically ill with a high fever. The differential diagnosis is extremely difficult in the postoperative period, as stated. Various postoperative complications, such as wound infection, breakdown of intestinal anastomoses, peritonitis, and intestinal obstruction, as well as such nonabdominal catastrophes as coronary thrombosis and pulmonary embolus, must all be thought of in a frequently severely ill patient going downhill rapidly.⁸ Probably the greatest aid to the clinician in making the diagnosis is simply realizing its possibility.

PROPHYLAXIS

A general measure to be considered under this heading is avoidance of the wholesale use of antibiotics for prophylactic purposes, especially in surgery. The realization that serious sequelae may follow prolonged antibiotic treatment should always be kept in mind. There is perhaps good indication to limit all antibiotic orders to a certain number of days when first written. Sensitivities of organisms at hand should be checked frequently. All bowel preparations should be kept short and intensive.² Probably the best combination preparation now at hand is neomy-

cin plus Sulfathaladine.¹⁹ The broad-spectrum antibiotics, such as Terramycin or Aureomycin, have long since been abandoned, fortunately, by most people working in this field. Some authors state that patients with known staphylococcal infections should be isolated and have special care for their dressings.⁹ Other staphylococcal foci of infections should be eliminated. Several authors have stated that cultures of the rectal contents should be taken preoperatively along with cultures from the bowel during the operation. They even feel that this may be an indication for specific antistaphylococcal therapy postoperatively if the cultural results are positive.

TREATMENT

Rapid consideration of the possibility that this disease may be causing the violent syndrome under observation is essential. The present antibiotics being given to the patient should be withdrawn. Heroic fluid replacement accompanied by electrolytes, plasma, and blood may be necessary. Many case reports were noted in which patients had lost in the neighborhood of 16,000 cc. of fluid in the stool per day. No special electrolyte problems were mentioned by the authors of these articles other than those which would usually be lost by severe diarrhea and vomiting. Hypochloremia and hypokalemia are probably the most common electrolyte problems. Levophed is also recommended by many authors, although the general consensus is that the most important replacement therapy of all is simply that of water and electrolytes. There is no substitute for close watching and supervision of the patient. If undiagnosed, the usual outcome is death in shock and renal shutdown within twenty-four to forty-eight hours.

The most highly recommended antibiotic in the articles read was erythromycin. Most patients were treated with this antibiotic, and the staphylococcus, at least four to five years ago, was practically always sensitive to it. However, it has been generally noted that the staphylococcus is developing resistance to erythromycin at about the same rate that it developed resistance to penicillin. Therefore, the outlook for erythromycin therapy in 1959 is considerably less bright than when most of the articles were written. Turnbull²⁴ states that the most effective drugs in his experience have been erythromycin, carbomycin, chloramphenicol, neomycin, and novobiocin. He has used erythromycin in a dosage of 500 mg. intravenously followed by 250 mg. every sixth hour. He has also used 750 mg. of novobiocin intravenously every eighth hour with a rapid reduction to 500 mg. every

eighth hour as soon as the clinical picture has definitely improved. The drugs may also be used orally, depending upon the patient's clinical status.

The method of Dr. Wendell Hall of this hospital, used in the case reported here, is noteworthy for its simplicity and effectiveness. He advises 8 gm. of Neomycin orally in one or two divided doses; then all antibiotics are stopped. It is hoped that this will rid the gut of most of the staphylococci, and the coliform organisms then will have a chance to regrow.

Sometimes, combination therapy is indicated. Prohaska's dramatic results with the use of ACTH are again to be cited. After 4 successive deaths, 3 patients survived after treatment with 20 mg. of ACTH three times a day for four to ten days. Cortisone has been used by other authors, too, but no specific enthusiastic endorsement has been given.^{23,25,26} The replacement of the bacterial flora of the bowel has been attempted by the use of Lactobacillus capsules. A recent innovation in therapy will probably not get the award as the most esthetic therapeutic venture of the year, but it may well rank as one of the most effective. This is a report by Eiseman and associates²⁷ on the use of normal fecal enemas as an adjunct in the treatment of pseudomembranous enterocolitis. They reported dramatic results in 4 cases with proved staphylococcus enteritis. Of these, 3 were postoperative and 1 was not. Within twenty-four to forty-eight hours, their patients responded dramatically from a very serious clinical picture that previously had not been reversed by heroic antibiotic and cortisone treatment. The staphylococcus was noted to disappear from the colonic contents within a day or a few days after the administration of a saline suspension of normal feces as a retention enema. They suggested that more widespread use of this adjunct, or even purifications of it for the more esthetically minded, should be tried in the future. They even suggested the oral use of fecal capsules for involvement of areas of the bowel above the colon.

CASE REPORT

History: R.M., a 33-year-old engineer, noted the rather insidious onset of chills and fever in early February 1959. A temperature of 103° F. was noted. There were no specific symptoms beyond a mild sore throat and some generalized myalgia. The patient's symptoms gradually improved, and his temperature dropped to normal. He returned to work on February 8, but his symptoms became worse and his temperature recurred. At this time, he noted some left-sided pleuritic chest pain. He reported to this hospital and was admitted on February 13.

Past medical history revealed that the patient had had

malaria while he was stationed in the Philippines in World War II. In 1946, he had epigastric pain and several emeses, and an upper gastrointestinal series was done at the University of Minnesota Health Service. He was told that it was negative. In 1949, he was hospitalized at the Health Service for eleven days for a fever of undetermined origin. His temperature then was as high as 104° F. Symptoms were of generalized malaise, again with no specific etiology or diagnosis. Penicillin allergy apparently developed at this time. There has been no history of alcoholism. The patient's wife and mother stated that he had had several episodes of unexplained abdominal pain with occasional emeses. They also stated that he frequently ran a high fever, as high as 105° F., with few other specific symptoms.

In May 1958, he was treated by a family physician for one of these episodes. The family doctor thought that he had a rather resistant case of sinusitis. He was treated with oral antibiotics and recovered in about a week. The patient himself denied or minimized the symptoms that had been described by his wife and mother.

Physical examination: The patient had a sallow, rather pasty looking complexion. His vital signs were normal and he was afebrile. Examination of his throat was negative. There were scattered medium moist rales in the left lower lung field posteriorly and laterally. No abdominal pathology was noted. The rectal examination was negative, as was the remainder of the physical examination.

Laboratory: Chest x-ray showed a left lower lobe bronchopneumonia. Urinalysis was negative. White count was 13,400 with 76 per cent polymorphonuclear leukocytes and 8 per cent eosinophils. Hemoglobin was 14.8 and the sedimentation rate, 51. Heterophil titer was 1:7. Cold agglutinins were 1:4 and, when the test was repeated eleven days later, they remained the same. Sputum and throat culture were both reported as beta hemolytic streptococcus. Blood culture was negative. Three stools for ova, parasites, and blood were negative. Carotene loading test was normal. Alkaline phosphatase, thymol turbidity, Bromsulphalein, serum cholesterol, and bilirubin were all normal. Zinc turbidity was slightly elevated at 12.8 units. Complete blood count was repeated a few days after admission and was normal, except for showing 9 per cent eosinophils.

Hospital course: The patient was treated with 500 mg. of oral Ilosone every six hours, beginning on February 13. He gradually felt better, and a roentgenogram and physical examination revealed that his pneumonitis had cleared. He remained afebrile. However, on February 24, the patient noted a severe epigastric pain and began to vomit. Questioning revealed that he had had mild epigastric distress for the past few days. Two loose stools were passed which were normal in color, but one which I viewed appeared to contain shreds of tissue. Examination revealed marked epigastric tenderness. Bowel sounds were decreased and high pitched. The chest was clear. Examination was otherwise not remarkable, aside from a temperature of 102° F. Blood pressure and pulse were normal. Roentgenograms of the chest and abdomen were essentially negative. White count was 12,100, with 87 per cent polymorphonuclear leukocytes. A stat urine amylase was normal. A smear of a rectal swab showed mixed organisms. The patient was started on nasogastric suction and intravenous fluids. He was seen by surgeons for an apparent acute abdomen, and they recommended that an emergency gastrointestinal study with Gastrografin be done. This did not show any perforation of the upper gastrointestinal tract. The temperature rose to 104° F., and the abdomen be-

came silent. It was decided to discontinue the Ilosone and continue symptomatic treatment until morning. The patient looked essentially the same the next day, and the fever continued to rise, reaching a high of 106° F. orally. About this time, hemolytic staphylococcus negative coagulase was reported growing out of a rectal swab obtained the night before for culture. Sensitivities done later on this revealed susceptibility only to penicillin, neomycin, novobiocin, Oleandomycin, and Kantrex. It was resistant to the broad-spectrum antibiotics and erythromycin.

The patient was treated with a stat oral dose of 4 gm. of neomycin. This was repeated once again in the morning. By mistake, he was also given 1 gm. of chloromycetin intramuscularly in the evening. Antibiotics were stopped after this. The patient's vital signs remained stable. His course gradually improved, and his fever dropped to normal by February 28. Ice packing was required over a considerable period of time. He had a few loose stools and a few emeses, but obvious fluid loss was never any great problem. A stool culture on February 25 was negative. However, a rectal swab culture again grew hemolytic staphylococcus. After that time, all stool cultures and rectal swabs except one were negative for staphylococcus, although normal coliform organisms did not appear, either.

Urine amylase and serum bilirubin began to rise on February 26. Urine amylase got as high as 1,031 units per hour on March 2. At this time, the bilirubin rose to its height of 2 mg. per cent. An alkaline phosphatase on March 4 was 33 cathode units. Complete blood count remained essentially normal. On March 3, the patient's temperature rose to 103° F. and was accompanied by a chill. Rectal swab done on this date again revealed a positive culture for hemolytic staphylococcus. Repeat roentgenograms of the abdomen and chest revealed no change. The patient's fever resolved spontaneously, and he did very well thereafter. His urine amylase and serum bilirubin dropped to normal, and he remained afebrile. He became asymptomatic and was discharged.

SUMMARY

The pathologic diagnosis of pseudomembranous enterocolitis and its clinical manifestations has been explored. A specific etiology, staphylococcal bowel infection, has been primarily dealt upon. The place of this specific etiology in the overall spectrum of the disease has been considered. The clinical problem at hand and current concepts about its solution have also been presented.

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Transthoracic Nephrectomy

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APPROACH to the renal fossa via the chest cavity is not new, for it was used in Great Britain as early as 1920 for casualties of World War I. During the past twelve years in the United States, this approach to renal lesions, more especially those requiring nephrectomy, has received considerable impetus.

In 1922, Fullerton, reporting in the Queen's Registry on a summary of British Army urinary organ wounds, was the first to stress the value of approaching the kidney through the chest. In 1930, Constantini and Bernasconi, reporting in the French *La presse médicale*, detailed a report of nephrectomy in a patient with severe kyphosis and attendant chest deformity. The procedure, which otherwise would have been difficult or impossible through the usual lumbar extraperitoneal route, was accomplished with no difficulty whatsoever. For the next two decades, surgery on the stomach, esophagus, adrenal, spleen, and gallbladder was sporadically performed via this route, with very satisfactory results.

In May 1946, Henry Mortenson,¹ prominent Australian urologic surgeon, reported a secondary nephrectomy via the chest for a hypernephroma previously declared inoperable after exploratory operation and subsequent x-ray therapy.

O'Connor and Head² in 1949 reported the ready removal transthoracically of tuberculous pyonephrosis in a kyphotic patient.

It remained for Clute^{3, 4} of Boston to publish in 1949 the first comprehensive report on the transthoracic approach detailing the case histories of 60 patients. Leadbetter⁵ of the Massachusetts General Hospital followed in 1958 with a series of 164 miscellaneous cases involving renal and adrenal pathology and also retroperi-

toneal node dissection for testicular malignant disease.

Specifically, the advantages and indications for this surgical approach are [1] wider exposure of pedicle area, [2] less manipulation of parts to be removed, [3] ease of securing major vessels, [4] ready access to nodal dissection, [5] rib cage deformities, that is, scoliosis and kyphosis, and [6] extensive lumbar scarring from previous surgery or x-ray therapy.

CASE REPORT

With the last indication in mind, the history of a 57-year-old woman will be reported in moderate detail. Mrs. W. F. was admitted to hospital June 25, 1957, because of urinary frequency, nocturia, and intermittent nausea and vomiting of one year's duration. Physical examination revealed a well nourished, middle-aged woman. Moderate tenderness was found over the left costovertebral angle, and 2 extensive lumbar scars were seen on the right loin. Her past history revealed 2 operations for stone in the right kidney eighteen months and three years previously. After the second operation, a nephrostomy tube had been utilized, and, after its removal postoperatively, the wound continued to drain for eight to nine months. Urinalysis revealed gross infection with culture positive for *Aerobacter aerogenes*, maximally sensitive to Furadantin. Blood urea nitrogen was 21.6 mg. per cent. Pyelograms and bladder films revealed a large staghorn calculus occupying the entire collecting system on the left and multiple small calculi on the right, with a contracted soft tissue renal silhouette (see fig. 1).

Blood calcium and phosphorus studies were negative. Subsequent pyelograms revealed moderately good function on the left and none on the right. The patient was discharged from the hospital on full doses of Furadantin and told to return in three to four weeks. Upon reentry to hospital, the patient's symptoms of infection had entirely cleared up and she stated she had not felt so well in years. Reluctantly, she accepted advice for the necessary surgery. Accordingly, she underwent extensive nephrolithotomy for the

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Fig. 1. Intravenous pyelogram done for a 57-year-old woman patient shows a large staghorn calculus on the left, multiple small calculi on the right, and a contracted soft tissue renal silhouette.

removal of her large, left staghorn calculus; the procedure was completely uneventful, and she was able to leave the hospital in twelve days, with the wound well healed and urine clear. The patient was readmitted eight weeks later for surgery on the right functionless calculous kidney.

The transthoracic route was elected because of the extensive lumbar scarring from the 2 previous surgical interventions on that side. With a consulting thoracic surgeon in attendance, the patient was given endotracheal anesthesia; a generous elliptical incision was made on the right chest wall, directly overlying and following the course of the tenth rib. Involved muscles were divided using meticulous hemostasis. With Alexander and Hedblom periosteal elevators, the entire tenth rib was mobilized. The rib was then resected after division of the periosteum. The pleura was opened and the lung on the right side exposed. This was packed off with wet lap sponges and retracted superiorly. Next, the diaphragm was incised generously in the direction of its insertion on to the chest wall (inferior side of eleventh rib). The right kidney was then exposed after insertion of a Finocchetti rib-spread-

ing retractor. Gerota's fascia consisted entirely of densely indurated fat. The renal pedicle was readily mobilized, doubly clamped, cut, and ligated with No. 2 catgut with transfixion sutures above and ligation sutures only below the proximal clamp. The ureter was identified and disposed of in routine manner. The kidney was removed, and sulfa powder was placed in the renal fossa; a Penrose drain was placed through a stab wound lateral to the line of incision. The chest closure was accomplished in anatomic layers; a No. 20 Foley catheter was placed in the chest cavity with instillation of streptomycin and penicillin before closure. Postoperative condition of the patient was completely satisfactory. The pleural suction tube was removed on the second postoperative day and the Penrose drain on the third day. The wound healed per primam, and sutures were removed in six days; the patient left the hospital on the eighth day.

This woman has continued to feel exceptionally well, with uninfected urine and complete absence of all symptoms. Basojel, salicylates, and a urinary antiseptic were continued for two months after her discharge. She sought medical advice eighteen months later because of a persistent, low grade abdominal pain, which was diagnosed as a duodenal ulcer. The lesion subsequently responded to dietary management. No recurrence of infection or calculi has been noted at six-month recheck examinations for the past two and a half years.

SUMMARY

1. A case of bilateral nephrolithiasis with a functionless right kidney, in which transthoracic nephrectomy was utilized, has been presented.

2. Contrary to the opinions of some surgeons, the liver in no way compromised the exposure obtained on the right side in this procedure.

3. This exposure, requiring a thoracic surgeon consultant initially, is relatively simple and with minimum experience becomes readily available to urologists interested in mastering a simple, extremely applicable technic.

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Congenital Hemolytic Anemia

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IN THE 18th century, Francois Voltaire commented of the Holy Roman Empire that it was "neither holy, nor Roman, nor an empire." In the same way, we may look at congenital hemolytic anemia—it is not always known to be congenital, hemolysis is not always evident, and the anemia is frequently neither evident nor severe. Even in a fully developed picture, thalassemia, sickle-cell disease, spherocytosis, and the hemoglobinopathies qualify as familial maladies which cause anemia by hemolyzing blood. Today, we wish to speak of the disease most commonly meant by the name "congenital hemolytic anemia." Like one of the FBI's 10 most wanted men, it goes by a number of aliases, describing several of its features and obscuring others. "Congenital hemolytic jaundice," "familial hemolytic jaundice," "acholic jaundice," "congenital hemolytic anemia," "familial hemolytic anemia," and "spherocytic," or "globe-cell" anemia have all labeled the same disease complex. "Hereditary spherocytosis" probably comes closest to characterizing it, and this name we will use here.

ETIOLOGY

The disease complex was first described by Vainlair and Masius,¹ in 1871, who drew a remarkably accurate illustration of the globular erythrocytes in "la microcythemie." In 1893, Wilson and Stanley² recognized anemia and splenomegaly as a part of the disease. In 1900, Minkowski³ gave the disease its classic description; and Chauffard,⁴ in 1907, showed the osmotic fragility of the red cells, completing the disease description. Probably the most complete recent survey of the disease lies in Dacie's book on the subject.⁵

FEATURES OF THE DISEASE

The disease as classically described is characterized by an anemia of variable amount accompanied by hemolysis and splenomegaly. A family history is generally present, and examination

of the blood shows a peculiar spheroid shape of the erythrocyte with increased susceptibility of the red cell to hemolysis in hypotonic saline solutions.

History. Chronic weakness is probably the most common complaint, although the patient may frequently have no symptoms or be unaware of the mild disorders accompanying his anemia. He may state that he has had unexplained mild anemia for much of his life. He may have had many episodes of icterus, and splenomegaly may have been noticed on previous examinations. In patients beyond adolescence, symptoms of gallstones are common and leg ulcers occasionally occur. Often, the younger patient may complain of the symptoms of a hemolytic crisis, with sudden onset of weakness, pallor, and icterus.

Physical findings. As compared with other hemolytic anemias, physical findings may be remarkably absent in hereditary spherocytosis. Icterus may be present, although it is nearly always mild. Of 19 adult patients of Young and associates, 6 never had clinical jaundice. It rarely occurs before the patient reaches school age and often occurs only in crises. Nearly all patients are said to have splenomegaly, the weight of removed spleens averaging 820 gm. in 10 of Young and co-workers'⁶ adult patients. A pale appearance and other findings referable to anemia may be present in some patients, although a fairly large number of those with spherocytosis are not significantly anemic.

Laboratory findings. Anemia is variable but not usually severe, the hemoglobin generally lying between 7.5 and 14 gm.,⁵⁻⁷ although more severe anemia may develop in crises. Nine of Young and associates' 28 patients had little or no anemia when initially examined. Reticuloocytes are usually numerous, ranging up to about 17 per cent in 2 series^{6,7} and averaging 14.6 per cent in 34 of Dacie's⁵ cases. Higher values are occasionally found, demonstrating the great erythropoietic activity present despite only moderate anemia.

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The most constant abnormality lies in the construction of the erythrocytes. They present an abnormal shape as seen on smear, initially described as "microcytes" but later being recognized to be spheroidal rather than disk-shaped. Because of this, the diameter of the cell is decreased and the total amount of hemoglobin per cell is increased, with a somewhat elevated mean corpuscular hemoglobin concentration. An interesting feature of the spherocytic red cell, to be elaborated later, is its increased susceptibility to hemolysis in hypotonic solutions. The mechanical fragility of this spherocyte is increased significantly over the normal cell, and, after incubation for one or two days at body temperature, suspension of the cells shows a marked tendency to lyse spontaneously. The serum bilirubin is mildly elevated.⁵⁻⁷ In 19 cases studied by Young and associates,⁶ the total bilirubin varied from 0.5 to 5.7 mg., averaging only 1.9 mg. per cent. and 4 patients never had values greater than 1 mg. per cent. Nearly all were in the indirect-reacting fraction, the one-minute determination always being normal. Urobilinogen is rarely found in the urine but is markedly elevated in the stool, as would be expected from the increased blood destruction. Values from 136 to 2,475, averaging 900 mg. per day, were found by Watson⁸ in a series of 10 cases.

Course of the disease. The typical course of the disease shows a constantly elevated rate of hemolysis throughout the patient's life. The production of red cells is invariably increased, although the bone changes often found in other diseases with increased erythropoiesis are seldom found in spherocytosis. The erythropoiesis and hemolysis generally stabilize at a hemoglobin level which remains fairly constant in any patient except for periods of crisis but varies considerably among patients. This status quo is occasionally interrupted by the crises characteristic of the disease. These crises are episodes of acute anemia and increased hemolysis, often accompanied in the unfortunate patient by fever, abdominal pain, increased splenomegaly, and systemic signs. The cause of these is rather obscure. They are generally believed to represent episodes of bone marrow depression which follow other systemic infections. In support of this, Battle⁹ cited a number of cases having diminished reticulocytosis during an acute crisis; as the reticulocyte count returned to normal, the crisis subsided.¹⁰ Other authors,¹¹ however, found an increased reticulocytosis, decreased cell survival time by radiochromium studies, and evidences of increased hemolysis during a crisis. These values returned to the patient's normal

after the symptoms of the crisis had passed.

Biliary lithiasis is very common. The stones are typical pigment stones which arise from the long continued high urobilinogen excretion. In Young and associates⁶ series, 12 of 14 patients past childhood had choledithiasis demonstrable by roentgenogram. Another finding less constantly reported is leg ulceration, similar to varicose ulcers. The etiology of these lesions is unknown. There is no known cause here for intracapillary occlusion as there is in sickle-cell anemia. Other associated anomalies, such as mental retardation and infantilism, have been reported sporadically.⁵ In very rare cases, bone thickening may arise secondary to increased erythropoiesis.

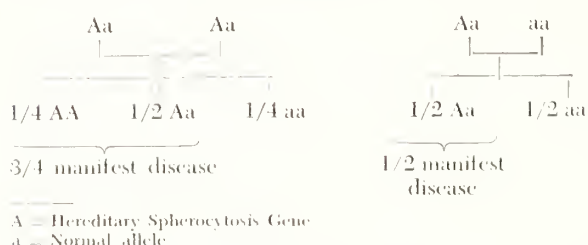
The mortality rate in spherocytosis is presumably low. The few reported autopsies show a normoblastic hyperplasia of the bone marrow, with a few sites of extramedullary hematopoiesis noted occasionally. The spleens are uniformly enlarged and show the same pathology as spleens removed at surgery. The weights of spleens reported range from 145 gm. to 1,190 gm. Dacie⁵ states that removed spleens generally weighed between 500 and 2,000 gm. Grossly, the spleens appear dark colored, firm, and congested but without enlarged hilar vessels. The architecture is not distorted, but the normal vessels, malpighian bodies, and trabeculae are separated by an engorged parenchyma. Section reveals a pulp characteristically packed with erythrocytes; the sinuses are often nearly empty. Some hemosiderin is usually present. Other organs are not generally affected.

Incidence. The disease may be found at any age. As would be expected in a congenital disease, some cases develop symptoms early, while other patients with less severe manifestations may be detected much later in life. Shapiro and associates¹² reviewed the literature in 1957 and found only 13 reported cases of hereditary spherocytosis diagnosed in the neonatal period. The detection of the remainder of cases seems to be distributed through various ages, with probably the majority of cases being detected in crisis in the first three decades. One case in Race's large series¹³ was that of a man who was found to be affected with the disease on an examination for bronchitis at age 77.

The over-all incidence of the disease is unknown. It is not considered to be rare and is said to be the most common of the hereditary hemolytic anemias, although only 8 cases have been reported in this hospital to date. It occurs with equal frequency in both sexes. The disease is chiefly found in persons of North European origin, although other racial stocks are also re-

ported with the disease. It is interesting to note that the neonatal case reported by Shapiro and co-workers¹² was a Negro child, and that of 13 adult cases with hereditary spherocytosis seen by them, 4 were Negro. It has been reported in Egyptians and in Filipinos.

The heredity of the disease has been studied intensively. The largest series was that of Race,¹³ who studied 138 members of 26 families in London. Data of other authors agree with his. The disease is felt to be inherited as a mendelian dominant, which would imply that at least one of an affected patient's parents must have the disease. It is also believed that persons found with the disease are heterozygous. The possible genetic matches are diagramed below:



The accuracy of a typical mendelian inheritance has been questioned by some findings, however. The incidence in children of *propositi* is about that expected, that is, about half the children of spherocytic parents have the disease, but only 20 per cent of siblings of *propositi* have been found to be effected. This discrepancy may rest on three factors: (1) incomplete case reporting, (2) incomplete penetrance of the gene, and (3) a rapid mutation rate. The latter is deemed not too probable, as the world would soon be peopled with spherocytic patients. The question of the possibility of homozygosity has been raised. The type of mating which could produce this was reported only once, between first cousins with the disease. Of this union, 3 children were affected, 1 was normal, and there were 2 miscarriages. Whether these were the expected homozygotes is unknown.

PATHOGENESIS

The majority of symptoms and findings in hereditary spherocytosis rest upon the destruction of erythrocytes long before their allotted four months. The study of the nature and destruction of these erythrocytes is the basis for elucidating the pathogenesis.

Methods. Before considering the specific abnormalities, it may be well to discuss briefly two of the methods used for estimating the survival and localization of erythrocytes: the Ashby differential agglutination technic¹⁴ or its modifica-

tions, and the more recent and familiar technic of radiochromium cell tagging. As used by the workers cited here,^{7,15} the older and more laborious Ashby technic consists of injecting donor cells of a different blood group into the patient. The cells in the sample to be analyzed are counted, the patient's cells are agglutinated by antiserum, and the remaining unagglutinated cells are recounted. The patient-donor cell ratio then can be calculated easily either in serum or in hypotonic saline solutions. It suffers the serious drawback that donor cells must be group O and the recipient, A or AB (M and MN subgroups have been used)¹⁶ and it cannot be used to identify the patient's cells within himself.

The cell survival method utilizing radiochromium was perfected in 1953 at the University of Chicago by Weinstein and LeRoy.¹⁷ This method "tags" any given cells, and the number of these cells mixed with any other cells can be determined by measuring the amount of radioactivity emitted by the isotope. The anticoagulated blood to be labeled by this technic is diluted and mixed with radioactive sodium chromate (Cr^{51}). The cells are then washed and injected into the recipient. The Cr^{51} is held almost entirely in the hemoglobin. It is best used to determine survival time and localization of destruction of the patient's own erythrocytes in his body.

Role of the spleen. The major problem in any hemolytic disease with splenomegaly is to determine whether the increased destruction is because of the large spleen or whether this large spleen is secondary to an intrinsic degeneration of the erythrocytes. Using the technics previously mentioned, one can (1) measure the survival of the patient's cells in himself, (2) measure the survival of normal cells in a patient with spherocytosis, (3) measure the survival of spherocytes in normal patients with intact spleen, and (4) measure the survival of spherocytes in otherwise normal patients without spleens. In addition, the survival of cells in isolated spleens can be measured.

All these studies have been carried out by various authors with highly consistent results. The cell survival time of spherocytic cells in patients with the disease is regularly shortened. Using the radiochromate technic, Coleman and Finch¹¹ observed a half-life of seven days; normal is about twenty-three days. Numerous other studies have been in this range. Normal cells injected into patients with hereditary spherocytosis, however, have a normal survival time, as shown by Dacie and Mollison,¹⁸ using the Ashby technic, and by others. Dacie and Mollison¹⁸ also transfused cells from patients with

hereditary spherocytosis into normal persons and found their survival time to be remarkably shortened. Emerson and associates⁷ repeated these experiments using the Ashby technic and found a half-life of about seven to ten days, with a total survival of thirty days; normal is about one hundred twenty days. They also transfused the same patient's cells into a normal donor whose spleen had been removed some time before, and the spherocytes survived normally. This illustrates that the spherocytes will survive normally in the absence of the spleen but that the spleen of patients with spherocytosis does not destroy normal cells.

The survival times found here might suggest that the spleen would function as a filter. Castle's group⁷ and Young's group¹⁵ preoperatively transfused patients with spherocytosis and with other diseases with normal cells and compared the concentration of these cells in the peripheral blood and in the excised spleen using the Ashby technic. Their results were as follows:

1. When other normal cells were transfused into a patient with thrombocytopenic purpura, the proportions of cells in the spleen and peripheral blood were the same.
2. When normal cells were transfused into a patient with spherocytosis, the patient had proportionately more of his own cells in his spleen than normal donor cells.
3. The osmotic fragility of spherocytes is much greater in the spleen than in the blood, whereas the fragility of normal cells is nearly the same in both.

Studies on freshly isolated spleens have been done by Young and associates.¹⁵ He perfused spleens through the artery with mixed populations of cells and analyzed the contents of the spleen, and of the effluent blood by means of the Ashby technic. In a control series, a spleen was perfused with two subtypes of group O blood, and the proportions were found to be nearly the same in the initial mixture, the effluent, and the splenic content showing that the spleen did not sequester nonhomologous normal cells. Using a mixture of spherocytes and cells from the patient, other spleens were perfused. Here, it was shown that the proportion of spherocytes was markedly decreased in the perfused blood and increased in the splenic pulp. Thus, the isolated spleens were seen to be capable of retaining the abnormal corpuscles and passing the normal cells. Schloess and associates¹⁹ and Fandl's group²⁰ counted the radiation over the intact spleen after autotransfusion with Cr⁵¹ labeled spherocytes and found the spherocytic cells to be concentrated there.

Role of the red cells. The characteristic feature of the red cell, spherocytosis, has been commented on previously. It is believed that this shape becomes more pronounced during the life of the cell, although even reticulocytes show some tendency towards spherizing. A sphere has the minimum amount of surface per volume of any shape, and therefore any expansion of the contents by osmotic increase in fluid results in rupture of the cell membrane. This shape is responsible for the osmotic fragility noted in the spherocyte. Fragility is generally demonstrated by measuring the hemolysis, that is, the amount of free hemoglobin, of cells in saline solutions of varying hypotonicity. This is reported in various ways. The most common routine report states the saline concentrations causing initial and maximum lysis, while another derives the saline concentration at which 50 per cent lysis occurs. The most accurate report, used here, graphs the per-cent hemolysis occurring at each of many saline concentrations. Several types of typical curves are shown in figure 1, which is taken from Young²¹ and exaggerated for demonstration.

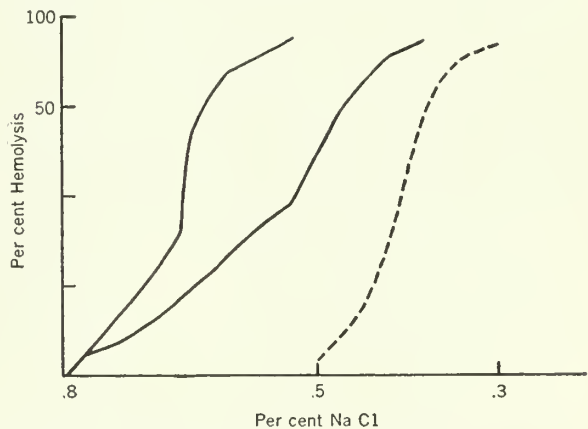


Figure 1. Graph showing per cent of hemolysis occurring at each of several saline concentrations.

Here, the per-cent saline solution is plotted as the abscissa and the per-cent hemolysis as the ordinate. The normal curve lies to the right, with hemolysis occurring at low tonicity. The curve to the extreme left shows a marked increase in hemolysis at higher tonicities, characteristic of severe spherocytosis. The curve between represents the "tailed" curve often seen in milder cases before splenectomy, with most of the cells nearer normal but a few cells very susceptible to lysis.

Other interesting phenomena of the spherocyte are found after incubation. The normal red cell will display slightly increased osmotic fragility after incubation at body temperature for twenty-

four to forty-eight hours. The spherocyte, however, markedly increases its fragility after this procedure. Even suspensions of cells in isotonic media will lyse markedly after incubation, a phenomena known as "autohemolysis." This tendency of the spherocyte can be slowed by addition of certain metabolites.

Spherocyte in the body. In the intact organism, the erythrocyte is never exposed to the osmotic concentrations causing hemolysis. The key to the in vivo hemolysis of cells may, however, rest with the autohemolysis phenomena. It has been shown above that spherocytes are sequestered in the spleen, probably for varying amounts of time. Young¹⁵ aptly characterizes these as "cells that stagnate in the backwater of the spleen." It has been demonstrated⁷ that cells in the spleen have a greater osmotic fragility than those in the blood. Immediately after splenectomy, with its attendant manipulation and splenic vasoconstriction, many abnormal cells are found in the blood, and the previous "tail" on the fragility curve is temporarily increased in length and height. Within a few hours, these highly susceptible cells vanish forever from the blood. Thus, sequestration and stagnation in the spleen seem to be a factor contributing to the cell degeneration, although these "aged" cells may be released from the spleen to be destroyed elsewhere later.^{21,22} An interesting light on this was recently cast by Motulsky et al.,²³ who transfused a patient, Pat, with spherocytosis with Cr⁵¹-labeled spherocytes from his splenectomized brother, Mike. The osmotic fragility of Mike's cells was observed to increase to the more fragile level of Pat's within a few days. The reverse, transfusing Pat's fragile blood into Mike, was also done, and the osmotic fragility was found to decrease to the more nearly normal level of the splenectomized Mike.

The peripheral blood in splenectomized patients shows a curve characteristically different from that before splenectomy.⁷ After the initial brief increase in abnormal cells described before, the "tail" of the fragility curve vanishes and the cells are of a more uniform nature, although their characteristically increased osmotic fragility still persists. This suggests that the more highly abnormal cells may result from the passage of the spherocytes through the spleen.

Metabolic abnormalities of the spherocyte. An immune mechanism of any type does not seem to cause the cellular abnormality, as the Coombs' test is almost invariably negative and no other antibody mechanism has been elicited. Young has done some interesting work on the metabolism of the spherocyte.^{21,24} He demonstrated that

the changes of autohemolysis were slowed by addition of glucose, mannose, or adenosine to incubated suspension of spherocytes, probably because the added substance aided in maintaining energy production. In studies with P³² uptake in incubated cells, his group showed that the uptake of phosphorus into adenosine triphosphate (ATP) and 2,3, diphosphoglycerate (2,3, DPG) was slow, and that inorganic phosphates inside the cells had high amounts of P³². This defect was converted to normal in a number of cases by incubating the spherocytes with adenosine. Young concludes from this that spherocytes may have an abnormal mechanism for the formation of inorganic phosphorus rather than the ATP pathway found in normal erythrocytes. He theorizes that the various defects in the spheroidal cell may stem from this defect in glucose metabolism. It is still not demonstrated, however, how this abnormality may effect the shape of the cell.

THE THERAPY

Splenectomy is the treatment of choice in hereditary spherocytosis and is therapeutically analogous in many respects to the treatment of myopia with glasses. There is little debate over the correct method of treatment; it results in immediate and dramatic improvement and relieves the symptoms while in no way affecting the primary disease.

Following splenectomy, the red cell count and hemoglobin rapidly return to normal levels, the reticulocytes decrease to normal, and the icterus and evidences of hemolysis vanish permanently. Hemolytic crises and anemia have not been observed postoperatively in follow-ups up to seventeen years.⁶ The osmotic fragility of the erythrocytes, however, remains permanently. Splenectomy is only contraindicated in early infancy, when an increased incidence of systemic infections has been observed.¹² Steroid therapy on a temporary basis has been attempted by Coleman and Finch at the University of Washington.¹¹ They treated 3 intact hereditary spherocytosis patients with large doses of cortisone or ACTH and found a rise in hematocrit and a fall in reticulocytes and fecal urobilinogen. In a fourth spherocytic patient who had a splenectomy, no change was observed during therapy. The authors concluded that the steroid effect was due to a nonspecific effect on the spleen, decreasing cell destruction or sequestration.

DIFFERENTIAL DIAGNOSIS

The features of the three main congenital hemolytic anemias are noted in the following chart. Differentiation in these presents little difficulty.

<i>Disease</i>	<i>Cells</i>	<i>Heredit</i>	<i>Primary race</i>	<i>Defect</i>
Hereditary spherocytosis	Spherocytes	Mendelian dominant	North European	Cell shape, glucose metabolism
Thalassemia	Platyctes "target cells"	Disease in homozygotes trait in heterozygotes	Mediterranean	Unknown
Sickle-cell anemia	Sickle-cells	Disease in homozygotes trait in heterozygotes	Negroes	Abn. hgb., change in cell shape with low O ₂

Acquired iso-immune hemolytic anemia presents the only other diagnostic problem in adults. A list of the characteristics of this disease which differentiate it from hereditary spherocytosis follows and is taken from Young and Miller.²⁵ These are ideal characteristics and may vary widely. Differentiation in many cases may be difficult without continued follow-up.

Iso-immune acquired hemolytic anemia:

1. Shows spherocytes only during crises, and the cells are normal during quiescence. The osmotic fragility displays the same variation.

2. The patients destroy normal cells as rapidly as their own.

3. The Coombs' test is positive in nearly all cases.

4. Thrombocytopenia is often present.

The differentiation is important here, as splenectomy is of variable value in cases of acquired hemolytic anemia. In the newborn, the differentiation between Rh iso-immunization and an early manifestation of hereditary spherocytosis may be extremely difficult.²⁶ In either case, exchange transfusions are often required to prevent the development of kernicterus in the child.

CASE REPORT

History. C.N., a 23-year-old farmer, was asymptomatic until December 1956 when he noted gradual loss of pep and left upper quadrant aching. He was more lethargic some days than others. He was admitted to an Air Force hospital where he was found to be mildly icteric and to have splenomegaly. His hemoglobin at that time was 15 gm. with a normal white count. His reticuloocyte count was 6 per cent, and bilirubin was 3.8 total. Bone marrow aspiration showed increased erythropoiesis with a myeloid-erythroid ratio of 1:1. Liver biopsy showed much hemosiderin. He was treated with cortisone at that time with no response and was told he had hemolytic anemia. He was discharged from the hospital, and his subjective condition remained unchanged and, indeed, has until the present time. He continued to have occasional episodes of fatigue, and his wife states that he often had scleral yellowing with these. He was admitted to Minneapolis Veterans Administration Hospital for diagnosis in April 1958. The findings at that time showed splenomegaly, a white count of 10,000, hemoglobin of 12.4 per cent with spherocytosis and poikilocytosis, reticuloeytosis of 13 per cent, and bilirubin of 0.3 direct and 2.4 total. A four-day specimen of fecal urobilinogen was 1,580 mg. per twenty-four hours. Os-

motie fragility screening test was positive. A cholecystogram was negative. He left the hospital at that time due to pressure of work before work-up could be completed. The same symptoms remained, but recently some right upper quadrant aching developed. He was readmitted to this hospital again on January 5, 1959 with essentially the same complaints.

Past history revealed only usual childhood diseases. He had no known episodes of jaundice, anemia, or weakness during childhood. His family history is interesting in that both of his parents have had gallstones. His father's were removed surgically. He has an uncle and an aunt on his mother's side and two brothers and one sister with diabetes. None of these have had known jaundice, anemia, or episodes of weakness similar to the patient's. His wife has never had jaundice or anemia. He has two children. His son, aged 27 months, has had no episodes of anemia but was found on examination to have moderate splenomegaly of 1 or 2 cm. His 7-month-old daughter was born with jaundice and evidence of splenomegaly. She was treated with two exchange transfusions at birth but has been anemic twice since. She has 3 to 4 cm. splenomegaly. A report from the Mayo Clinic states that the child has increased reticuloeytosis, spherocytosis, and increased osmotic fragility of her red cells. Their impression is that she has hereditary spherocytosis. The patient's wife's Rh type is unknown. He is Rh positive.

Physical examination. Physical examination revealed a well-developed, mildly obese male in no distress with a blood pressure of 130/75 and a pulse of 64. Questionable scleral icterus was present. There were two small left posterior cervical nodes which were palpable. There was a grade 1 to II apical systolic murmur with no cardiomegaly. The liver was felt 1 cm. below the costal border and was firm and nontender. The spleen was descended 4 cm. and, on inspiration, was nontender and firm. The rest of the examination was normal.

Laboratory studies. Laboratory studies showed a hemoglobin which varied from 12.2 to 13.8 on various examinations. Red count was 4.2 million, hematocrit, 38 per cent; mean corpuscular volume, 91; mean corpuscular concentration, 37; and mean corpuscular hemoglobin, 32. White count was 6,950, with a normal differential. Coombs' test and cold agglutinins and serologic reactions for venereal disease were negative. Platelets were 163,000, the sedimentation rate, 5 mm.; and reticuloeytes, 24 per cent. Liver function tests were normal except for a bilirubin of 2.6 total and 0.2 direct. Urinalysis was normal; a four-day specimen of fecal urobilinogen was 385 mg. per twenty-four hours. Osmotic fragility test showed hemolysis starting 27 per cent at 0.66 per cent sodium chloride and going to 94 per cent hemolysis by 0.57 per cent sodium chloride. The control started with 13 per cent hemolysis at 0.57 per cent sodium chloride and had 96 per cent at 0.48 sodium chloride. Chest x-ray was negative.

Hospital Course. The patient had a biopsy of the left posterior cervical node on January 21, which showed only chronic inflammation. Mild weakness has persisted. It is planned to remove the patient's spleen in the near future.

SUMMARY

Hereditary spherocytosis is a disease characterized by variable hemolytic anemia, splenomegaly, and an abnormal erythrocyte. The pathology of the disease may rest on an abnormality of glucose metabolism in the red cell.

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CHRONIC RENAL INSUFFICIENCY is often accompanied by anemia. Recent tests conducted at the University of California in San Francisco have led to the belief that cobaltous chloride therapy may be a valuable aid in correcting this condition. Of 12 patients treated orally with 100 to 200 mg. cobaltous chloride daily for approximately two months, 6 improved and probably required fewer transfusions; the renal disease did not appear to be affected. No goiter or hypothyroidism occurred as a result of therapy, and occasional moderate depression of radioiodine uptake disappeared rapidly after therapy was discontinued.

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Cervical Spondylosis (Osteoarthritis) Simulating Coronary Heart Disease

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THE DIFFERENTIAL DIAGNOSIS of chest pain is an important and sometimes perplexing problem. Because of the potentially serious nature of pain in the chest, the subject of its differential diagnosis is covered thoroughly in medical texts and is reviewed periodically in the literature. However, we feel that one entity has not been sufficiently stressed, although it was carefully described and reported in 1927 by Phillips¹ and has been emphasized on occasion since.²⁻⁵ Further, we think that this entity is quite common and can closely simulate coronary artery disease. This paper presents 10 patients who were seen in cardiac consultation with the suspected diagnosis of coronary artery disease but were found to have cervical spondylosis with radiular pain.

These 10 patients were seen during a period of eight months, reflecting the high incidence of this syndrome. Ollie⁶ in 600 consecutive thoracic pain problems diagnosed a radiular syndrome in 197 cases. Davis,⁷ who has written a very excellent monograph on this subject, feels that this syndrome is very common, that it can simulate coronary artery disease remarkably, and that, as awareness increases, the frequency of the diagnosis will increase accordingly.

The following case reports exemplify the characteristics and vagaries of this common syndrome.

CASE REPORTS

Case 1. H.E., a 55-year-old white unemployed male, was first seen June 25, 1958. History revealed aching precordial and left neck pain associated with excitement and exertion and moderate dyspnea on exertion of seven years' duration. A diagnosis of angina pectoris had been made at onset of illness. The patient had been taking a

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vasodilator since that time. He had not worked for one year and had been supported by welfare since then. Physical examination was within normal limits. The original impression of the examiner here was coronary heart disease. Routine laboratory work, chest x-ray, and electrocardiogram were within normal limits. A Master's test to the point of pain was performed, and, despite the fact that the patient had rather severe pain that persisted for about an hour, the electrocardiogram remained normal. It was then noted that he had left anterior chest wall tenderness and tenderness at the spinous processes of the lower cervical region. Cervical spine x-ray revealed minimal hypertrophic changes anteriorly and narrowing of the intervertebral foramina between C3 and 4 on the right and C6 and 7 on the left. Cervical traction treatments were prescribed which provided immediate relief of symptoms and complete relief of symptoms after one week of daily treatments. On follow-up visit nine months later, the patient still had only very rare stinging anterior chest pain for which he used cervical traction at home with complete relief of symptoms.

Case 2. O.B., a 45-year-old white male partsman, was first seen August 26, 1957. Four months earlier, while he was sitting in church, this patient had noted rather severe intra capsular pain associated with alternate hot and cold feelings. The next day, he had aching between his shoulders with some left anterior chest pain that radiated into his left upper arm. Deep breathing and coughing aggravated the pain and nitroglycerine was said to have produced some relief. He was hospitalized at this time elsewhere for three weeks and was discharged with the diagnosis of "mild heart condition." The patient stated that, nine months before his visit here, he had had a somewhat similar episode for which he received no medical attention. History was otherwise negative. Physical examination was within normal limits. Electrocardiogram and chest x-ray were normal. X-ray of the cervical spine revealed small marginal hypertrophic spurs at the posterior margins of the sixth and seventh cervical vertebrae with foraminal narrowing at this level bilaterally, most marked on the left. This patient was treated and reassured that he had no heart disease since he was asymptomatic at the time of his original visit. A follow-up letter written by the patient twenty-two months later stated that he had just occasional minor shoulder pains.

Case 3. R.E., a 49-year-old white male farmer, was first seen October 21, 1957. This patient complained of aching, burning, substernal and bilateral upper arm pain of one year's duration which came on with exertion and lasted approximately one minute. He had received a

diagnosis of coronary artery disease elsewhere and was applying for workmen's compensation for this reason. During the week before his visit here, he had awakened at night on several occasions with episodes of this pain. He also noted that cold weather and dampness caused him to have pain more frequently and, if he wore warm clothes around his neck, he obtained some relief. Nitroglycerine had produced no benefit. Physical examination was entirely within normal limits with no chest wall or spinal tenderness. The impression was noncardiac pain, possibly of radicular origin. Chest x-ray, electrocardiogram, and a Master's examination were normal. X-ray of the cervical spine revealed localized lipping of the interspace between C5 and C6 and slight narrowing of the intervertebral foramina on the right from C4 to C6. No other abnormalities were noted. After three weeks of cervical traction treatment, the patient had had almost complete relief of his symptoms. He was given a cervical traction apparatus and was to continue cervical traction at home. A follow-up examination six months later revealed that he then had no chest or neck pain of any kind.

Case 4. J.L., a 46-year-old white male farmer, was first seen October 16, 1957. History revealed dull, aching, right pectoral chest pain of eighteen months' duration which was produced by exertion and lasted ten to thirty minutes. The patient had noted that it was particularly likely to occur with use of the arms, as in digging, and that it was produced by running but not by walking. No relief had been obtained with the use of nitroglycerine. A diagnosis of coronary artery disease had been made elsewhere, and the patient had been using a vasodilator for one year. Physical examination was negative except for considerable tenderness in the right anterior chest and moderate tenderness over the fourth and fifth cervical spinous processes. Chest x-ray, electrocardiogram, and Master's examination were within normal limits. X-ray of the cervical spine revealed a sharp bony spur posteriorly at the inferior margin of the body of the fourth cervical vertebra, which extended posteriorly to impinge upon the neighboring foramen. A similar small spur was seen at the same location in the interspace above. The patient was started on cervical traction treatments, and his symptoms were first made worse by this treatment. Treatment was continued, however, with considerable benefit. Four weeks after the initial examination, the patient was entirely free of pain. He went back to working at hard manual labor and has continued to do so to the present time. He has continued to have, on occasion, mild chest pains that have responded quickly to cervical traction treatment at home.

Case 5. P.M., age 54, a white male carpenter, was first seen April 13, 1957. This patient had exertional, aching right pectoral and precordial chest pain of one year's duration. The pain usually lasted some minutes but occasionally lasted as long as several hours. Occasionally, it was sharp and stabbing, and, rarely, it occurred on the right side of the chest. He had noted that pain occurred less frequently after a good night's rest. He also had noted moderate dyspnea on exertion, orthopnea requiring 3 pillows, and a good deal of nonproductive cough since the onset of his disease. The patient was hospitalized at a veterans hospital shortly after the onset of his illness and received a diagnosis of coronary artery disease for which he was given a pension. His family physician had treated him on several occasions since with medications for heart disease. Physical examination was entirely within normal limits except for mild obesity.

SUMMARY OF CLINICAL DATA OF 10 MALE PATIENTS WITH CERVICAL RADICULAR SYNDROME

Case	Age	Occupation	Presenting diagnosis	Exertional	Pain—Location	Foraminal narrowing	Treatment	Response
1—H.E.	55	Unemployed	Angina pectoris—7 years	Yes	Precordial Left neck	Yes C3-C4 right C6-C7 left	Cervical traction	Excellent
2—O.B.	45	Partisan	"Heart condition"—4 months	No	Interscapular Anterior chest	Yes C6-C7 left	Reassurance	Good; occasional pain
3—R.E.	49	Farmer	Angina pectoris—1 year	Yes	Upper arms Substernal	Yes C4-C6 right	Cervical traction	Excellent
4—J.L.	46	Farmer	Coronary artery disease—18 months	Yes	Right pectoral	Yes C4-C5 right	Cervical traction	Excellent; occasional pain
5—P.M.	54	Carpenter	Coronary insufficiency—12 months	Yes	Right pectoral Precordial	Yes C6-C7 right and left	Cervical traction	Excellent
6—D.R.	63	Farmer	Coronary disease(?)—2 weeks	No	Substernal	Yes C6-C7 left	Cervical traction	Excellent
7—M.S.	66	Farmer	Hospitalized as possible myocardial infarction	No	Across anterior chest and both shoulders	Yes C4-C5 right and left	Reassurance	Good
8—L.M.	53	Farmer	"Coronary insufficiency"—1952. Hospitalized as possible myocardial infarction, 1958	No	Left pectoral	Yes C5-C7 right and left	Reassurance	Good; occasional stiff neck
9—J.A.	53	Farmer	Possible angina—1 month	Yes	Anterior chest	Yes C6-C7 left	Cervical traction	Excellent
10—W.L.	42	Farmer	"Rule out angina"—3 months	No	Left pectoral Left arm	Yes C4-C5 right	Cervical traction	Good

The original impression of the examiner was that organic heart disease was probably not present, but that a work-up was required. A chest x-ray revealed calcification of the aortic arch, but the heart size and contour were normal. Moderate osteophytosis of the lower dorsal spine was noted. An electrocardiogram was normal, and a Master's examination was negative. After this information was available, the patient was seen again. It was noted that compression of the patient's cervical spine produced his chest pain and that traction on the head in the sitting position immediately relieved it. It was also noted that there was some local tenderness about the fifth thoracic spinous process and that no chest wall tenderness could be elicited. A diagnosis of cervical spondylosis was made. X-ray of the cervical spine revealed a localized bony overgrowth posteriorly at about the interspace between C6 and C7, projecting into the intervertebral foramina bilaterally. Subsequently, he was started on cervical traction treatments that completely eliminated his symptoms; he has continued to use an overhead cervical traction apparatus at home. He was asymptomatic shortly after traction treatments were started and, when he was seen one year later, had had no recurrence of chest pain.

Case 6. D.R., a 63-year-old white farmer, was first seen September 16, 1957. This patient stated that two weeks before his visit here, in the evening after he had pushed a stuck ear, he experienced severe left chest and arm pain that lasted for three or four hours. Mild similar pains occurred on and off after that, and a day before admission the patient noted more severe substernal chest pain that lasted for fifteen minutes on each occasion and occurred repeatedly. The pain was associated with profuse sweating. He also noted that movement of the arms and riding on a tractor seemed to aggravate the pain, while walking had no effect. He was seen in the emergency room on the evening previously by a member of the house staff, who took an electrocardiogram and referred the patient with the tentative diagnosis of coronary artery disease. The patient recalled that two years previously he had had a similar episode that subsided without treatment. Physical examination revealed an obese white male but was otherwise negative. Chest x-ray was normal, as was an electrocardiogram and a Master's examination. X-ray of the cervical spine revealed marked narrowing of the interspace between C6 and C7 with marginal hypertrophic changes and evidence of disk degeneration. There were large marginal osteophytes projecting through the foramina on the left side. Treatment with cervical traction was initiated with complete relief of symptoms. Follow-up one year later revealed that the patient was continuing to use cervical traction treatments at home intermittently. He had had no severe chest pain since treatment was started.

Case 7. M.S., a 66-year-old white male farmer, was first seen on September 3, 1957. This patient gave a history of ten days' duration consisting of four- to five-minute episodes of dull precordial chest pain that radiated into both shoulders and down the left arm. It was not affected by activity but seemed to be made worse by driving a tractor. There was no dyspnea, perspiration, or other associated symptoms. The patient was originally seen at the clinic and was admitted to the hospital with the impression of possible angina pectoris. Physical examination revealed only a slight pectus excavatum. The following day, the additional history was obtained that, for one year, the patient had had some

stiffness of his neck, for which he had received physical therapy in the past with some relief. Examination of his cervical spine on this day revealed restricted lateral flexion and rotation with some tenderness near the second rib on the left at the costosternal junction. Routine laboratory work was normal as was the serum transaminase. Chest x-ray was normal, and an electrocardiogram was within normal limits except for some slight sagging of the S-T segments in standard limb lead I and fifth and sixth precordial leads. An electrocardiogram two days later was described as being without change. An x-ray of the cervical spine revealed a bony overgrowth posteriorly about the interspaces between C3 and C5, most marked at C4 with foraminal narrowing bilaterally at this level. The patient's symptoms subsided promptly, and he was not started on cervical traction. When he was seen in follow-up ten months later, he had had no recurrence of his chest pain.

Case 8. L.M., a 53-year-old white male farmer, was admitted to the hospital on March 3, 1958 with a suspected diagnosis of acute myocardial infarction. History revealed that, early in the morning on the day of admission, the patient awoke with dull, aching, left upper chest pain that persisted for one-half hour and then disappeared. The patient went back to sleep but was awakened shortly thereafter with a similar pain that was more severe and associated with profuse cold perspiration. The pain gradually increased in severity and continued until the time of admission. At the height of the pain, there was some radiation down the left arm. The patient stated that two years previously he had had a similar episode for which he had been hospitalized elsewhere for three weeks with a diagnosis of "acute coronary insufficiency." Five years previously, a diagnosis of "coronary insufficiency" had also been made for which hospitalization had not been advised. Systemic review revealed that the patient had had some trouble with posterior neck pain and sore shoulder muscles for six or eight months before admission. The examination revealed a blood pressure of 100/70 and mild perspiration. Physical examination was otherwise within normal limits. Electrocardiograms on March 3 and 4, 1958, were within normal limits, and the transaminase was normal. On the third hospital day, a chest x-ray was normal. A roentgenogram of the cervical spine revealed narrowing of the interspaces between C5 and C6 and C6 and C7, with posterior marginal osteophytes encroaching on the foramina at both these levels bilaterally. The patient's symptoms had entirely disappeared shortly after admission, and he was dismissed without cervical traction treatments. His discharge diagnosis was cervical spondylosis with chest pain due to the radicular syndrome. A follow-up eighteen months later revealed that chest pain had not recurred but nuchal stiffness had continued.

Case 9. J.A., a 53-year-old white male farmer, was seen in surgery clinic on January 24, 1958, because of low back and left leg pain. The surgeon arranged an immediate cardiac consultation because he had noted the following incidental history that he felt suggested angina pectoris. For one month, the patient had a "cramp" in the lower anterior chest which lasted two to five minutes and came with or without exertion. He also had a "soreness" in the upper dorsal region and marked nervousness with dyspnea and palpitations on exertion. Physical examination revealed tenderness in the upper dorsal spine, but no chest wall tenderness or other abnormal findings were noted. Chest x-ray was normal. Although a previous electrocardiogram showed nonspecific T wave changes, one taken during this exam-

ination was normal. X-ray of the cervical spine revealed narrowing of the interspace between C6 and C7 with foraminal narrowing bilaterally at this level. A diagnosis of the radicular syndrome was made, and cervical traction and diathermy treatments were begun. After 3 treatments, the chest pain had disappeared completely.

Case 10. W.L., a 42-year-old white male farmer, was first seen February 14, 1958. For three months, this patient had had dull, aching, left upper chest pain that radiated into his left arm and lasted for variable lengths of time. Excitement and fatigue seemed to initiate the pain, which was associated with a faint feeling and considerable apprehension. He was referred by his family physician because of the suspicion of coronary artery disease. Physical examination revealed moderate left anterior chest tenderness. Chest x-ray and electrocardiogram were normal, but x-ray of the cervical spine revealed some narrowing of the intervertebral foramina on the right between C4 and C5. A diagnosis of radicular syndrome was made. The patient was started on cervical traction treatments from which he obtained good relief. Cervical traction treatments at home were prescribed, but a follow-up letter eight months later revealed that the patient had had no further chest pain and consequently had not purchased a cervical traction halter. A follow-up letter from his family doctor almost two years later stated that symptoms had recurred and cervical traction was being initiated.

DISCUSSION

Some of the pertinent clinical data of the 10 patients presented above are shown in the table. It will be noted that all were males over 40 years of age and that the oldest was 66. It is probably important that most of these patients were accustomed to hard work, since 7 were farmers. This is reasonable inasmuch as men generally receive more trauma to their spine, either by accident or hard physical work. Since collecting these initial patients, however, we have observed the same syndrome in a few women and in some men who were white-collar workers. It is revealing that all 10 patients were referred for cardiac evaluation. Most of these patients were suspected of having angina pectoris, and 1 was hospitalized with the suspected diagnosis of acute myocardial infarction. The thoracic pain occurred at various sites and frequently radiated to the shoulders and arms. The typical bandlike pain of angina was not common but did occur and was indistinguishable from anginoid pain. Pectoral and precordial pain was the most common. Sweating, dyspnea, and other ancillary symptoms often occurred, making the clinical picture even more like coronary heart disease. Noteworthy is that half of these patients found that their particular discomforts were aggravated by some type of exertion. Generally speaking, a careful history differentiated this from the exertional pain of true angina pectoris, and the resemblance to effort angina pectoris was often superficial. Each of these 10 patients had posi-

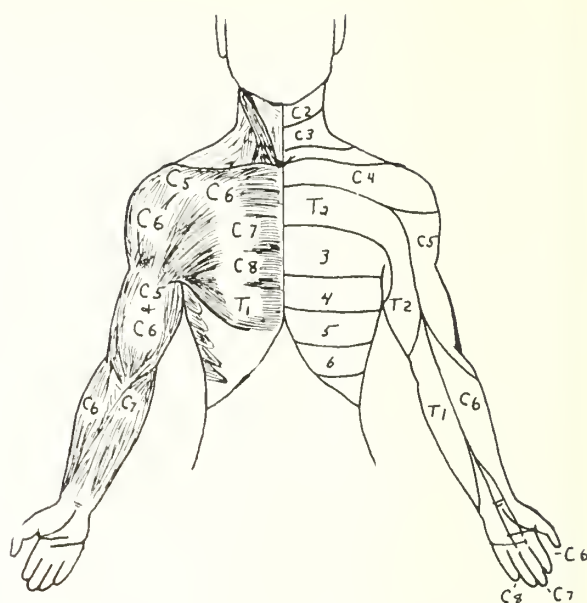


Fig. 1. Topographic distribution of dermatomes and myotomes. The myotomes are depicted on the reader's left. From Davis.⁷

tive x-ray findings, the most important of these being definite narrowing of the intervertebral foramina on the basis of osteoarthritis. The response to treatment in these patients was very gratifying and helpful in substantiating the diagnosis.

The characteristics of nerve root pain have been quite completely described. That irritation of the dorsal root causes a sharp, localized, shooting pain in the distribution of that dermatome is especially well known. These dermatomes have been mapped repeatedly by different workers with quite consistent agreement. These dermatome "maps" are included in many textbooks and are well stressed in medical teaching. Of somewhat more recent knowledge is another type of pain with a different radiation pattern which is produced by the stimulation or irritation of a partially diseased ventral nerve root. These areas have been called myotomes to distinguish them from the more well-known dermatomes. This mapping can be confirmed by the injection of hypertonic saline into the region just lateral to the interspinous ligament.⁸ Such injections will produce a dull, aching, poorly localized pain in an area corresponding to the myotome of that spinal segment. Figure 1 shows the topographic differences between the dermatomes and myotomes of the cervical and upper thoracic spinal nerve segments. This, then, is the explanation of how pathologic changes in the upper spine can produce anterior chest pain that may resemble

the poorly localized and "pressing" pain of coronary insufficiency.

The pathologic basis of radicular pain has been extensively described. Irritation of nerve roots may be produced by disk protrusion, neoplasms, posterior osteophytes encroaching upon intervertebral foramina, fibrosis and hypertrophic changes of the ligamentum flava, and fibrosis and constriction of the nerve root sleeve. By far the most common cause of radicular pain originating in the cervical spine is osteoarthritis with posterior osteophytes encroaching upon the intervertebral foramina. Whether the initiating lesion of this disease occurs in the disk or in the facet is not entirely clear. If foraminal narrowing is the chief factor, extension of the spine, which further narrows the foramina, is likely to produce symptoms. However, if root sleeve fibrosis is the most prominent pathologic change, flexion of the spine with upward traction on the spinal cord may produce symptoms. Because of these many possible pathologic changes, almost any spinal motion or spinal position in the individual patient can be a precipitating cause for symptoms.

Any or all of the fibers of either the ventral or dorsal root may be irritated. Pain then may occur in any part or all of the dermatome or myotome or both. Pain is produced on certain motions of the spine or in certain positions of the spine, and, characteristically, relief can be obtained by other spinal positions. In individual patients, these positions and motions can be of any variety. Especially important diagnostically are the results of the compression test. This is performed simply by pressing firmly down upon the erect head of the sitting patient; this maneuver usually causes neck pain and, frequently, chest pain. Relief of symptoms by manual traction on the head is of equal diagnostic importance. Déjerine's sign, or exacerbation of symptoms by coughing, straining, and sneezing, occurs in approximately 25% of patients with the radicular syndrome. Radicular pain commonly has its onset after prolonged recumbency. Characteristically, it comes on in the early hours of the morning; relief is obtained by changing the position of the spine such as by sitting up or turning over in bed. The fact that symptoms should be produced by prolonged resting in bed is not surprising, since the position of the spine while the patient is recumbent in a soft bed is quite different than any position the spine assumes while the patient is erect during the day. That radicular pain often appears during prolonged recumbency is especially important to appreciate, for this could readily be confused with so-called angina decubitus.

Spinal tenderness is practically constant in patients who have radicular syndrome. This valuable sign is elicited simply by pressing firmly on each spinous process in turn. Tenderness of the chest wall is another valuable sign and is usually present when sought. It may be anywhere in the thoracic region but is usually parasternal or precordial. Many tense individuals will have upper spine or thoracic wall tenderness, but, with experience, one can usually differentiate these patients from those with the cervical radicular syndrome. For example, the tenderness in the anxious individual is usually more variable, bizarre, and widespread.

The important physical signs of this syndrome, therefore, are anterior chest wall tenderness, spinal tenderness, production of symptoms by various spinal positions and motions, and relief of symptoms by other spinal positions and motions. The cardinal symptom of coronary artery disease is anterior chest pain associated with exertion. Since patients with radicular pain so commonly have the onset of their symptoms while working, it is simple to conclude that the symptoms are "exertional" and only by a careful history can the differentiation be made. Patients with cervical radicular pain very commonly have symptoms while walking, and it is uncertain why

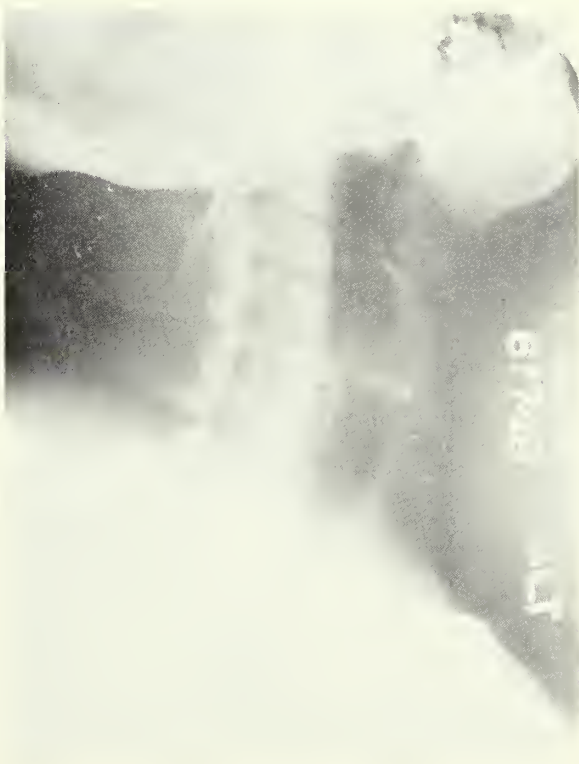


Fig. 2. Left oblique view of normal cervical spine.

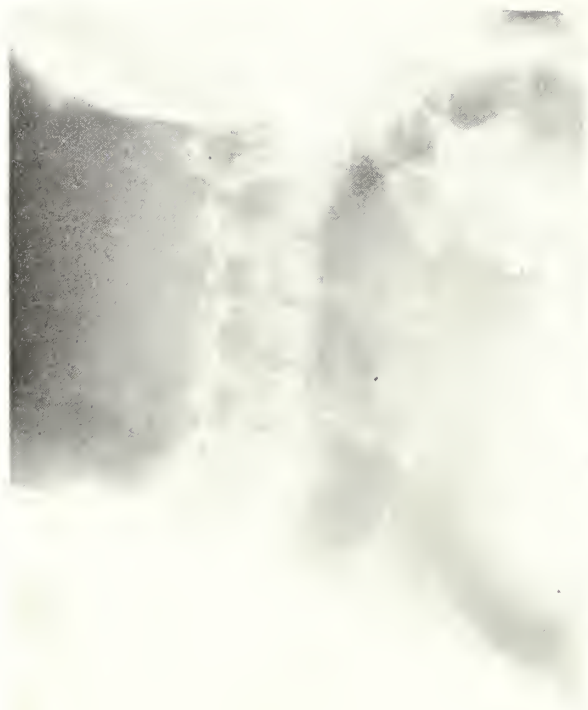


Fig. 3. (Case 5) Left oblique view of cervical spine showing localized bony overgrowth posteriorly at the interspace between C6 and C7 projecting into the intervertebral foramen.

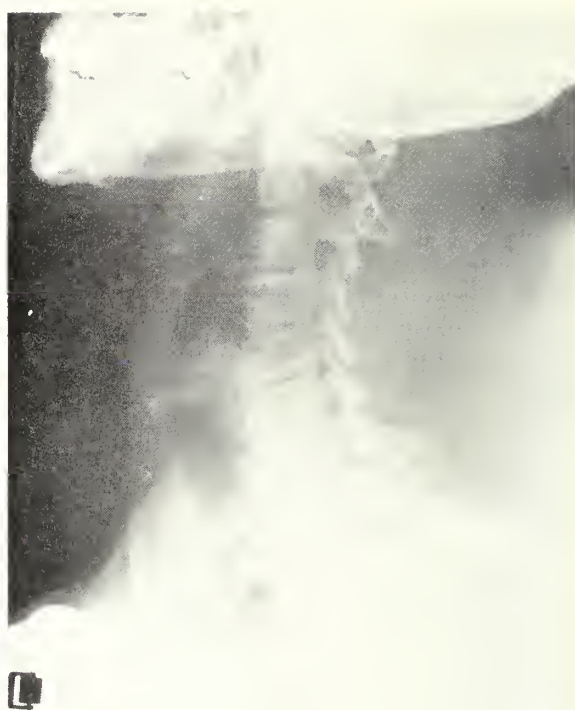


Fig. 4. (Case 8) Left oblique view showing narrowing of the interspaces between C5 and C6 and between C6 and C7 with posterior marginal osteophytes encroaching on the foramina.

this should be so. An explanation might be that a certain slouch or thoracic kyphosis is present in many people when they walk, or the intermittent jarring that is transmitted to the spine with each step might be the explanation. Symptoms also commonly occur while the patient is driving a car or tractor, working over a desk, reaching up, and working in a bent-over position. Nowhere in clinical medicine is the patient's history more crucial than in the differential diagnosis of thoracic pain. Indeed, the only reliable means we have to diagnose angina pectoris is by taking a careful history and by listening to the patient tell his story. Similarly, the patient with the cervical radicular syndrome, if he is allowed to talk, will usually tell the doctor that his pain comes from the upper spine. For example, a farmer may say that the only time he has the pain is when he drives his tractor over rough terrain, or a housewife may have the pain only when she irons clothes or hangs out her wash. Even though the pain may come on while walking, a patient may notice that altering his posture will relieve the distress. This type of in-

formation comes to the physician only if he takes a careful history.

The roentgen findings are frequently helpful in the diagnosis of this syndrome. Evidence of cervical or upper dorsal osteoarthritis, particularly when intervertebral foraminal narrowing is present, provides additional evidence in favor of this diagnosis. However, unless oblique views of the cervical spine are taken, narrowing will often be missed. It must be remembered, however, that many individuals have rather marked roentgen changes of the upper spine and have no symptoms. More important is that patients with this syndrome may have very few roentgen findings. Important soft tissue changes can occur and are just as capable of causing nerve root irritation as bony changes. This also explains why some patients may have a distribution of pain that does not correspond with the spinal level of visible x-ray changes. Figure 2 shows the roentgen appearance of a normal cervical spine in the left oblique view. Figure 3 illustrates the typical appearance of localized cervical spondylosis and encroachment of the inter-

vertebral foramen. Figure 4 is another example of the typical roentgen appearance in this syndrome. The physical findings and the patient's story, therefore, are more decisive in making the diagnosis of a radicular syndrome than the x-rays alone. It is very probable that upper dorsal spine changes are responsible for many instances of chest pain, but this area of the spine is particularly difficult to examine by x-ray and the intervertebral foramina at this level are difficult to portray by x-ray.

The treatment of thoracic pain arising from upper spine spondylosis is usually simple and gratifying. Cervical traction with simultaneous diathermy seems to be most effective. Cervical traction is done with the patient sitting, utilizing a simple head halter and initial weights between 10 and 15 lb. This is done daily for twenty to thirty minutes with increasing weight according to tolerance and effect. If the pain is due to cervical spondylosis, the patient usually will note partial or total relief after two or three days. Most patients will need periodic traction, and a simple halter device can be rigged up at home for convenience.

A few pitfalls should be mentioned. We have had the opportunity to see some patients who have both coronary heart disease and the cervical radicular syndrome. In these persons, it is usually not too difficult to differentiate the symptoms and signs and allow one to suspect that both conditions exist. That coronary heart disease may masquerade itself in many atypical ways is well known, and many of these patients deserve a thorough cardiovascular work-up to exclude coronary heart disease even though cervical spondylosis exists. Other causes of thoracic pain must be excluded. It is not within the scope of this paper to discuss the innumerable causes of chest pain, but the prime importance of a careful history cannot be overemphasized. One must also be on guard that the cause of radicular pain is not a serious lesion, such as a primary or metastatic tumor, in spite of the presence of osteoarthritis in the spine.

If this paper has a *raison d'être*, it is to emphasize the importance of the cervical radicular syndrome in the differential diagnosis of chest pain that may be confused with coronary heart disease. Every physician has seen the sorrowful example of the patient with the misdiagnosis of coronary heart disease. He often becomes a "noncardiac" cardiac cripple—afraid to move, afraid to work, afraid to live. Not only is the label of coronary heart disease difficult to eradicate from the patient's mind when once made,

but the entire future of that patient's life is often irrevocably altered. For example, once a patient has the label of coronary heart disease, it is often impossible for him to obtain life insurance at a normal rating in spite of subsequent negations of the original diagnosis. In this regard, it is regrettable that frequently the diagnosis of coronary heart disease is made or implied by minor, nonspecific changes in the electrocardiogram alone. The number of persons that have been literally crucified on inverted T waves must be large, indeed! Likewise, there are undoubtedly many individuals with the cervical radicular syndrome who have received the erroneous diagnosis of coronary heart disease.

The features enumerated above, especially a careful history and physical examination, will usually allow the physician to make a correct differential diagnosis. In discussing the differential diagnosis of thoracic pain, Dry⁹ aptly summed it up when he said, "Despite all the helpful data which can be derived from the various diagnostic procedures, there is no alternative to skillful interrogation of the patient."

SUMMARY

A total of 10 patients with cervical spondylosis and chest pain are presented. The so-called cervical radicular syndrome may closely resemble the clinical picture of coronary heart disease. The character and location of chest pain may be quite similar, and the importance of differentiating these 2 common conditions is emphasized. Particularly important and rewarding in the differentiation are a careful history and physical examination. The clinical, pathophysiologic, roentgenologic, and therapeutic features of this syndrome are presented.

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John Silas Lundy, M.D.

J. ARTHUR MYERS, M.D.

IN DECEMBER 1957, attention was called to a fine honor recently accorded Dr. John S. Lundy. He had been elected first president of the Anesthesia Memorial Foundation, an organization dedicated to helping young men in anesthesiology and to building the specialty. Since that time, he has continued to edit the Section on Pain which has appeared quarterly in *THE JOURNAL-LANCET*. This section was introduced in November 1952. As Dr. Lundy was retiring from the clinical staff of the Mayo Clinic, the high regard in which he is held in Rochester and his numerous contributions to the benefit of humanity everywhere were recorded in the September 26, 1959, issue of the *Rochester Post-Bulletin*. Editor Charles Withers has kindly given permission to reprint the following:

"Dr. John S. Lundy, internationally known specialist in anesthesiology and founder of the Section of Anesthesiology at the Clinic in 1924, will retire October 1. He will leave Rochester and continue the private practice of his specialty in Chicago.

"Dr. Lundy was born in Inkster, North Dakota, July 6, 1894. As a boy 5 years old, he began to take a lively interest in medicine and determined to become a specialist in medicine. He received the degree of Bachelor of Arts in 1917 from the University of North Dakota and that of Doctor of Medicine from Rush Medical College of the University of Chicago in 1919. In 1919 and 1920, he was an intern in the Harper Hospital in Detroit, and, from 1920 to 1924, he was engaged in the private practice of medicine in Seattle.

"On April 1, 1924, at the invitation of Dr. W. J. Mayo, Dr. Lundy came to Rochester as head of the Section of Anesthesiology at the Mayo Clinic. He founded the Section and directed it until 1952, when

he became a senior consultant. He was appointed an instructor in anesthesiology in the Mayo Foundation in 1925, an assistant professor in 1928, an associate professor in 1931, and professor in 1934. He established the first laboratory of gross anatomy to be used at the Mayo Clinic.

"As a result of his extensive original researches in anesthesiology and the volume of anesthesia required at the Mayo Clinic, Dr. Lundy early became recognized as an international authority in this field of medicine. In addition, he was a pioneer in the graduate training of young physicians in anesthesiology at the Mayo Clinic, and he was one of the founders of the American Board of Anesthesiology, Inc., which sets the standards and conducts the examination of physicians who wish to become specialists in that branch of medicine. In 1942, he served as president of that board. He was certified as a specialist in anesthesiology by that board in 1938.

"Dr. Lundy established the first blood bank in the United States at the Mayo Clinic in 1935. In 1923, he developed the specifications for and was responsible for the construction of a four-control machine for the administration of anesthetic gases; between 1922 and 1932, he developed an apparatus for artificial respiration for surgical patients; and, in 1925, he devised a special syringe and equipment for local anesthesia. His theory and practice of "balanced anesthesia," which he introduced in 1925, is widely known. Recently, with the new resources of drugs which are antagonists to certain other drugs, he evolved a refinement known as "precision balanced anesthesia." In 1932, he introduced a solution of procaine anesthesia for use in spinal anesthesia, and, in 1934, he contributed to the advancement of intravenous anesthesia by demonstrating the ad-

vantages of several new barbiturates when they are employed intermittently with intravenous anesthesia.

"On June 18, 1934, he introduced the use of Pentothal Sodium, an intravenous anesthetic agent now used in surgery all over the world. On March 17, 1942, he opened the first postanesthesia recovery room in the world at St. Mary's Hospital, embodying a practice currently in use throughout the United States and Canada. He is recognized as an authority on the mass treatment of casualties in time of disaster.

"Many honors, national and international, have come to Dr. Lundy for his pre-eminence in his field. In 1943, the Hahnemann Medical College of Philadelphia conferred upon him the honorary degree of Doctor of Laws, and, in 1948, the University of North Dakota awarded him the honorary degree of Doctor of Science. In 1948, North Dakota presented him with an honorary license to practice medicine in that state, the first such license ever issued, so far as is known; and, in the same year, he was the recipient of the distinguished service medal of the American Society of Anesthesiologists. He is licensed to practice medicine in the states of Illinois, Washington, Nevada, Texas, Minnesota, and North Dakota.

"Dr. Lundy currently is president of the Anesthesia Memorial Foundation. He served as president of the American Society of Anesthesiologists in 1946, and he has been chairman and secretary of the Section of Anesthesiology of the American Medical Association. In 1931, he was made an honorary member of the Grand Forks District Medical Society, and, in 1938, he was made an honorary member of the Omaha Midwest Clinical Society. In 1940, he became a nonresident member of the North Dakota Academy of Science, and, in 1941, he was chosen an honorary charter member of the Michigan Society of Anesthetists.

"He is a member of the Cuban National Society of Anesthesiology and the Société Française d'Anesthésie et Analgésie. He is a corresponding member of the Sociedad de Estudios Clínicos de la Habana and a corresponding fellow of the Association of Anaesthetists of Great Britain and Ireland. He is also a member of the Minnesota State Medical Association, the Minnesota Society of Anesthesiologists, the American Society for Pharmacology and Experimental Therapeutics, the International Anesthesia Research Society, the Associated Anesthetists of the

United States and Canada, the Alumni Association of the Mayo Foundation, the Society of the Sigma Xi, the Beta Theta Pi academic fraternity, and the Alpha Kappa Kappa professional medical fraternity. He is an honorary fellow of the International College of Surgeons.

"In 1955, Dr. Lundy was appointed a member of the medical advisory committee of the American National Red Cross. He is a consultant to the surgeon of the United States Fifth Army and a consultant in anesthesiology to the Shriners' Hospitals for Crippled Children. He has been president of the Rochester Board of Public Health and Welfare and, until August 1959, was a member of the Mineral Springs Sanatorium at Cannon Falls. He is also a member of the board of directors of the Olmsted County Tuberculosis Association.

"During World War II, Dr. Lundy was a national consultant in anesthesia for the Wartime Graduate Medical Meetings, a government project, held in Washington, D. C. He also served as a civilian consultant in anesthesiology to the Office of the Surgeon General, United States Army, and he was a member of the Subcommittee on Anesthesia of the National Research Council in Washington, D. C.

"In April 1957, he was appointed a contractual consultant to the Great Lakes Hospital of the United States Navy, and, in company with a number of other outstanding specialists, he lectured on anesthesiology at various Army and Navy medical installations in Hawaii, Guam, and Japan.

"Dr. Lundy is the author of *Clinical Anesthesia*, the first authoritative volume in the field, published in 1942, and he has contributed more than 600 papers on anesthesiology and related subjects to the medical literature.

"Dr. Lundy was married to Miss Lenore Mittelstadt, of St. Paul, on September 5, 1925. Dr. and Mrs. Lundy have three children: Richard Allen of Chicago, Joan Lenore of Philadelphia, and John Charles, a student of languages in the University of North Dakota."

Here is another example of a world-famous physician whose accomplishments are shared by North Dakota and Minnesota.

It is pleasing to state that Dr. Lundy will continue to edit the Pain Section in THE JOURNAL-LANCET from his office at the Veterans Administration Research Hospital, 333 East Huron, Chicago 11, Illinois.

Book Reviews

Bleeding Esophageal Varices. Portal Hypertension

H. R. LEIBOWITZ and L. M. ROUSSELOT, 1959. *Springfield, Ill.: Charles C Thomas. 986 pages. \$24.50.*

This is a wonderful source book which will prove of great interest and value to anyone interested in the problem of portal hypertension, whether he be pathologist, physiologist, or clinician. The monograph contains 31 chapters, all but 2 of which concern examples bearing upon the complex problem of the nature of portal hypertension, the origin of esophageal varices, and clinical aspects of hemorrhage occurring in patients with either intrahepatic or extrahepatic portal vein obstruction. The last 2 chapters of the book deal with the surgical management of portal hypertension.

The seriousness of the problem posed by the patient with hepatic cirrhosis with varices is evident from Leibowitz's statement that, of 50 such patients observed by him, 44 or 88 per cent were dead within a year from the onset of the first hemorrhage.

The authors favor conservative control of bleeding from esophageal varices by luminal compression with an indwelling inflated balloon. By the application of a special headress worn by the patient they have been able to extend the usefulness of the Sengstaken-Blakemore tube. However, this writer would be inclined to believe that use of local esophageal and gastric cooling will come to displace tamponade achieved through intraluminal pressure in the arrest of hemorrhage from esophageal varices.

Every aspect of the complicated problem presented by the patient with portal hypertension, whether owing to hepatic cirrhosis or to an extrahepatic block of the portal vein, is presented. Moreover, the authors have had a wide and long experience in this field, and in a sense this important book could be termed a summary of their experience. It obviously is by no means a hasty effort. In fact, all facets of the discussion reveal evidences of a mature and realistic appraisal of the multifaceted nature of portal hypertension. Certainly everyone who has an interest in the general problem of portal hypertension will want to have ready access to this useful book.

There is much to praise and little to criticize in this important work. Undoubtedly it will continue for many years to be the Bible and chief source of reference for all who have an interest in the vexing problem of portal hypertension and its management.

OWEN H. WANGENSTEIN, M.D.
Minneapolis

Total Surgical Management

JAMES D. HARDY, M.D., 1959. *New York: Grune & Stratton, Inc. 292 pages. Illustrated. \$9.50.*

This monograph was written for the general surgical practitioner as well as for senior medical students, interns, and surgical residents. In the words of the author, "the main aim has been to detail points in everyday sur-

gical practice." The title implies a counterpart of partial surgical management, and this is a bit confusing. Words are loosely used by the author, as indicated by the following quotations, "The History *Itself* . . . Every item of possibly useful information regarding the present complaint will have been woven into a *fabric* which one hopes will provide a *mosaic* of the disease. One *hallmark* of the competent physician is the care, perception, and accuracy with which he performs and records clinical examination. Few other *facets* of patient care are so easily attainable and so often ignored as is the orderly head-to-toe physical examination" (reviewer's italics). The Oxford student might expect more precise diction in a treatise on science.

The following excerpts indicate the character of this book, "When empyema does form it must be evacuated. This objective may occasionally be achieved with simple needle aspiration or with closed catheter drainage but open thoracotomy and decortication will often be required." "A precise preoperative cardiac diagnosis is absolutely indispensable for routinely satisfactory cardiac surgery. With a poorly placed chest incision and erroneous information regarding the intracardiac pathology, the operation will result in frustrated and ineffectual effort in an inexcusable number of instances. Again, the diagnosis is achieved by various combinations of clinical evaluation, the electrocardiogram, roentgenographic studies that may include angiocardiology, and cardiac catheterization. Only occasionally is 'exploratory cardiotomy' justified."

The book covers a wide variety of subjects from "head to toe." The discussion of each subject is necessarily brief, and some of it is in outline or cookbook form. It may be valuable to the student or intern but is probably of little value to the experienced surgeon. Each chapter has references, many of which are of recent publication. The author refers to recent literature. This book contains much that is obvious. It is easily read and probably serves a useful purpose as an elementary outline of surgery.

CLAUDE S. BECK, M.D.
Cleveland

Canadian Cancer Conference, Volume III

B. W. BEGG, editor, 1959. *New York: Academic Press, Inc. 461 pages. Illustrated. \$12.00.*

The title of "Canadian Cancer Conference" may be misleading, since this volume is a collection of 28 contributions written and co-authored by 18 Canadians, 18 Americans, 6 Englishmen, 1 Australian, and 1 Swede. Thus, it truly represents an international symposium on nucleic acids, genetics, viruses, and tumors and briefly touches on the biology of cancer in broader terms.

The preface modestly states that the purpose of the series is to "give a broad picture of cancer research to Canadian scientists working in that field." In effect, the 3 volumes of the series are to be recommended to work-

ers in cancer research everywhere, and the National Cancer Institute of Canada deserves commendation for having made them available to the scientists of all nations.

Volume I of the series covers discussions on experimental tumors, tumor-host relationships, enzymes, and ionizing radiations. Volume II concerns the cell, leukemia, chemotherapy, hormones, and cancer and basic mechanisms. The present Volume III is divided into sections on nucleic acids, genetics, viruses, and tumors and biology of cancer.

The complexity of the subject matter renders classification difficult, and it is not clear, for example, why a chapter by Barnes and co-workers on murine leukemia and the radiation chimera belongs in the section on viruses or why papers on the effects of radiation were grouped with F. M. Burnet's discussion on the biology of cancer. These are, however, minor shortcomings. A more regrettable editorial decision was the omission of any open discussion of the papers which we assume must have taken place at these meetings. The editors of the Ciba Symposia have demonstrated that inclusion of carefully edited discussion remarks can greatly contribute to the value of this type of symposium, and it is to be hoped that others who publish proceedings of meetings will adopt this method of life-reporting.

Even without this added material, the present book presents the reader with 461 pages which are well indexed; richly documented with references; and generously illustrated with tables, charts, and halftones. The contributions are well written and up-to-date. New fields such as problems of gene control, protein and nucleic acid synthesis, localization of newly formed nucleic acids, enzymatic synthesis of polynucleotides, interaction of genes and carcinogen in leukemogenesis, nature and biology of the SE polyoma virus, and the cytopathogenicity of RNA-rich particles from human leukemic and tumor cells make fascinating reading.

It is inevitable that any group of studies that "penetrate into the utmost recesses of cellular chemistry" encounter more questions than they produce answers, for such is the nature of this enigmatic disease.

Those working in this difficult field of cancer research as well as those in related areas of biologic research will do well to make this third volume of "Canadian Cancer Conference" part of their libraries. They may not read it from cover to cover in one sitting, but they will return to it often for information, stimulation, and intellectual pleasure.

FREDDY HOMBURGER, M.D.
Cambridge, Massachusetts

A Primer of Water, Electrolyte, and Acid-Base Syndromes

EMANUEL GOLDBERGER, M.D., 1959. *Philadelphia: Lea & Febiger.* 322 pages. \$6.00.

This book is designated by the author as a primer. As such, it contains elementary material on such subjects as the conversion of milliequivalents to milligrams, the meaning of milliosmols, and the composition of extracellular and intracellular fluid and of various special body fluids. In addition, it contains listings of various types of replacement solutions; practical considerations for administration of parenteral fluids; and diet lists which assist in the planning of low-protein, low-potassium, and low-phosphorus diets.

The theoretic considerations presented in the book have all been previously presented in other books and syllabi of this type. Dr. Goldberger utilizes the Brøn-

sted terminology in lieu of the more confusing "fixed acid and base" concepts, which makes this a somewhat more clearly read book than some of the older "primers." The clinical part of the book also offers little material not presented previously by authors such as Daveport and Welt.

From the standpoint of the pediatrician, one would wish that Dr. Goldberger had ignored considerations of fluid therapy in children. His handling of this subject is extremely superficial and occasionally in error. For instance, on page 274, the water requirements are given as 100 to 300 ml. per kilogram of body weight for children up to 22 kg. Use of the larger figure might be tolerated but would certainly place a strain on the homeostatic mechanism of any child ill enough to require parenteral fluid therapy, and even the smallest figure is in excess of that recommended generally for the older children in this weight group. On the next line, 14 to 23 ml. per kilogram of body weight is not equitable with 30 to 50 ml. per pound, and one wonders which the author is proposing. On page 275, hypertonic saline is probably indicated in the treatment of water intoxication caused by over vigorous treatment of hypernatremia rather than calcium gluconate. Too little emphasis is placed on the necessity for administering fluids to children on a twenty-four-hour basis, on the dangers of hypodermoclysis, and on the necessity for treating small burns (5 per cent and over) in young children.

This book will probably be of little or no value to the pediatrician, internist, or surgeon conversant in any degree with modern concepts of fluid therapy. It may be of value to those with limited information in this field who would like a volume with information as to elementary mathematical concepts, clinical classifications, and practical replacement solutions under one cover. There is a bibliography at the end of each chapter which the interested reader can pursue for more detailed information.

ELEANOR COLLE, M.D.
Minneapolis

The Surgical Treatment of Scoliosis

LOUIS A. GOLDSTEIN, M.D., 1959. *Springfield, Ill.: Charles C Thomas.* 100 pages. Illustrated. \$6.75.

Scoliosis has been a challenge and a stimulus to orthopedic surgeons for years. In spite of numerous investigations, there remains much that is unknown about this disease and a great many patients are still treated under the diagnosis of idiopathic scoliosis.

"The Surgical Treatment of Scoliosis" is a short, concise résumé of the entire subject with an analysis of the author's results in 54 cases. It is possible that this material could have been condensed even more and reported in a surgical journal rather than being published as a bound manuscript.

The subject of scoliosis is large. Other surgeons have reported larger series than the one contained herein and have contributed more original work.

The material is well presented and well illustrated. The modes of therapy outlined are well accepted. The average physician does not relish treatment of the scoliotic patient and probably would rather refer him to the orthopedic surgeon. The orthopedic surgeon would find this to be just another review without any recent additions. The student and the resident surgeon, however, would find it helpful.

W. B. STROMBERG, JR., M.D.
Chicago

BOOK REVIEWS—(Continued)

Early Diagnosis

HENRY MILLER, M.D., *Editor*, 1959. *Baltimore: Williams & Wilkins Co.* 375 pages. \$6.50.

In this book, 25 British physicians successfully collaborate in a discussion of early diagnosis. In a modest format, these authors knowingly scan the presenting features of many common ills. Here we find the signs and symptoms confronting the general practitioner from day to day.

Without art, a discussion of these familiar subjects could be trivial. When they instruct, old and wise doctors are likely to remind us of the art as well as the science of medicine. Before the laboratory can speak a categorical and sometimes equivocal yes or no, the doctor must see, hear, and touch. At this time he practices the art of medicine.

"Early Diagnosis" is largely concerned with this art. Genuine art and common sense usually achieve a close alliance. This concert is present in the book we are talking about. One finds the flavor of it in, for example, Dr. Ian Aird's discussion of alimentary malignancies. He says that, if barium meals were consumed every six months by all subjects beyond the age of 50, one would now and then discover an unsuspected cancer. Then he remarks that "the cost of discovering one carcinoma of the stomach in this way might well amount to a quarter of a million pounds for each positive radiologic diagnosis." A man who speaks in this fashion has something profitable to say.

We think that all the authors of "Early Diagnosis" have spoken profitably and well. In spite of a diverse authorship, the book manages a uniform level of clear and wise medical writing. There are very few pomposities, such as "armamentarium," or offensive etymologies, such as "broad spectrum." One suspects that these good medical writers contrived their best with the counsel of Dr. Henry Miller, editor and physician in neurology at the Royal Victoria Infirmary in Newcastle on Tyne. We commend his effort and invite your pleasure as reader.

HARRY SOFORKENKO, M.D.
Los Angeles

Anesthesia: A Manual for Students and Physicians

STUART C. CULLEN, M.D., 1958. *Chicago: Year Book Publishers, Inc.* 295 pages. \$5.50.

The first edition of this well-known work appeared in 1946. The current edition is the fifth, and the author has tried to bring his text up to date. He has had an opportunity, during the revisions of the previous four editions, to evaluate his presentation, and he has arrived at some rather definite ideas. It is very difficult for the author of any medical work to ensure that what he presents is up to date because progress in the introduction of new techniques, drugs, procedures, physical agents, and analgesic agents is so rapid that what is written may be incomplete before the volume is off the press. The new antagonistic drugs, for example, have opened several avenues to the anesthesiologist who seeks precision in general anesthesia, as every devoted anesthesiologist should. Yet even more will be forthcoming before the antagonists currently available are adequately described.

The book is indexed and is printed on good paper. Evidence of the industry of the author is impressive.

JOHN S. LUNDY, M.D.
Chicago

Orthopedic Nursing

MARY POWELL, S.R.N., 1959. *Baltimore: Williams & Wilkins Co.* 464 pages. \$6.50.

This text for nurses is now in its third edition since 1951. This does reflect its popularity among nursing institutions. The book is concise and readable. The description of splints and appliances is, on the whole, excellent; however, there is a tendency to illustrate and stress the old standard appliances dating from Sir Robert Jones and to forego the newer developments in brace manufacturing. Some of the braces shown are of obviously poor construction. The Milwaukee brace for scoliosis, for example, is shown as a poorly fitted travesty on the beautifully constructed and fitted brace now being made in Milwaukee.

Many chapters are spent on skeletal tuberculosis. The author used this disease to illustrate technics in nursing care applicable to other orthopedic problems involving bed care with immobilization. One cannot help but feel, however, that tuberculosis is overemphasized.

Many of the illustrations are those from the text on fractures by Sir Reginald Watson-Jones. Manipulative reduction of vertebral fractures is stressed. Such manipulative reduction of cervical fracture dislocations would not be favored by many orthopedic surgeons in this country.

This book illustrates the differences of opinion in orthopedic thinking. It will serve well as a very readable supplement to similar texts on orthopedic training.

JOHN MOE, M.D.
Minneapolis

Pathogenesis and Immunology of Tumors

G. V. VYGONCHUKOV, M.D., *Editor; translated by R. CRAWFORD*, 1959. *New York: Pergamon Press.* 258 pages. \$12.50.

This book is not of interest to the general practitioner. It is rather a book of interest to researchers in the field of cancer and virology. The book is dedicated to the honor of L. A. Zil'ber and consists of papers by various authors, most of whom have been his students and associates.

The premise of the book is that viruses are probably the causative agents of cancer in man, and certainly in animals, either with or without the presence of a carcinogenic stimulus. In substantiation of this conclusion, numerous experiments are reported in some detail. The Shope papilloma of rabbits, the Brown-Pierce rabbit carcinoma, the breast cancer of mice, and fowl leukosis factors were extensively investigated in these experiments. Some experiments using tumors of human origin are also reported.

The development of antigens from both animal and human tumor material is described in some detail. These antigens are believed by the authors to be related to the virus causing the tumor and to be type specific.

Immunity, antibody formation, and vaccination were also used in attempting to prove the thesis of viral origin of tumors. Animals desensitized to protein extracts from normal tissue were found to show a specific reaction to protein extracts of certain tumors, and antibody formation was demonstrated in animals using tumor extracts as antigens.

Your reviewer found that his basic knowledge of these procedures was insufficient to allow him to judge critically the validity of the conclusions drawn.

HAROLD W. MORGAN, M.D.
Mason City, Iowa

(Continued on page 35A)

BOOK REVIEWS

(Continued from page 138)

The Physician and the Law

ROWLAND H. LONG, LL.B., LL.M., 1959. *New York: Appleton-Century-Crofts, Inc.* 296 pages. \$5.95.

This book, as stated in the foreword, is "primarily intended for the instruction of medical students and physicians." In 277 pages of text, it covers the broad fields of relationship of physician to patient, physician to courts and rules of law, insurance for doctors, and so on. Of course, in covering so much material in such a limited space, the book must of necessity be almost in outline form and difficult to digest mentally when taken in large doses. This is not the kind of book one can skim through hurriedly; it is the type of book from which a doctor can read selected sections at leisure times.

Since this book is designed for study over a period of time by students, it is best utilized for such extended absorption. Nevertheless, it will benefit the practicing physician to read the illustrative instances of malpractice; the procedure for commitment of the mentally ill; the rules covering artificial insemination, adoptions, abortions, and revocation of license to practice; and other subjects which are well expressed and clearly paraphrased and indexed.

Occasionally the author lapses into incomprehensible legalistic jargon such as "The res includes the attending circumstances, and the application of the rule presents principally the question of the sufficiency of circumstantial evidence to justify the jury in inferring the existence of the principal fact in issue, namely, the defendant's negligence." Fortunately, such lapses into jargon are rare, and, for the most part, the material is set forth in lucid and interesting fashion.

In order to guide their actions, the physician and the student, who must of necessity concentrate on the science of medicine, need to understand contract rules, legal standards which measure medical care, and the relationship of medical science to civil and criminal law. This book includes such rules, standards, and relationships and is thus recommended as a valuable resource for medical practitioners and students.

SIDNEY LORBER, LL.B.
Minneapolis

Work and the Heart

FRANCIS F. ROSENBAUM, M.D., and ELSTON L. BELKNAP, M.D., Editors, 1959. *New York: Paul B. Hoeber, Inc.* 362 pages. Illustrated. \$12.00.

This is a most unusual and timely monograph. It brings the reader in a concise and readable form the most recent information relating to work and stress and strain on the heart. The material has been derived from the first conference concerning this subject held in Wisconsin. Seventy-four outstanding authorities in the various disciplines that relate to the subject participated in this conference, including internists, cardiologists, biophysicists, industrial surgeons, and lawyers.

The monograph discusses many of the problems and social implications of heart disease as it relates to work. The quality of the manuscript is excellent, and the format and illustrations are very well done. The editors, Rosenbaum and Belknap, must be congratulated, along with their associates, for having furnished to the medical profession a complete and well-documented series of manuscripts pertinent to this subject. It is true that

many problems are left unsolved and that many new problems arose as a result of the conference, but, nevertheless, this is one of the most worthwhile contributions we have had concerning the cardiac in industry.

The book could well be read by every practicing physician, but certainly it is a must for those who are interested in cardiovascular disease and occupational medicine.

JOHN F. BRIGGS, M.D.
St. Paul

Experimentation in Man

HENRY K. BEECHER, M.D., 1959. *Springfield, Ill.: Charles C Thomas.* 74 pages. \$3.50.

This short monograph, originally appearing in the *Journal of the American Medical Association*, sums up briefly and quite clearly the author's titled subject.

Dr. Beecher brings together the thoughts and comments of many other authors and experts in this field in a single volume which should be read by all persons engaged in that area of medical research that requires the "ultimate experimental animal."

There are several sections in Dr. Beecher's book that I feel require special attention. In the section of ethical and moral aspects, I do not think sufficient stress is laid on the contemporary nature of our prevailing attitudes toward human experimentation. It is probably true that they are derived from the Judeo-Christian heritage of Western man. However, these viewpoints are quite recent and are based more on current social, uniquely democratic interpretations rather than on any great basic moral truth that has been recognized as such through the ages.

In the chapter entitled "Legal Considerations," insufficient stress is placed on the actual jeopardy in which the investigator, utilizing human subjects, may be placing himself. Rather than a few mild statements about the law not keeping up with current advances and mention of the case of Slater vs. Baker in 1767, it would be well to state more flatly, as was said at the National Institutes of Health by one of the legal advisors, "I presume that you are on the fringes of medical knowledge. You certainly are on the fringes of the law." This is an area that is causing as much if not more concern than any other at the present time, since 2 or 3 adverse court decisions could place in jeopardy a large part of the clinical research being conducted in this country at the present time.

In the section on codes, Dr. Beecher summarizes quite well the general opinion concerning human experimentation as expressed in various codes. I do not think that he gives sufficient space to the fallacies of these documents. Nazi physicians could have claimed they satisfied the provision in the postwar "Nuremberg Code" that "the experiment should be such as to yield fruitful results for the good of society, unprocurable by other methods or means of study, and not random and unnecessary in nature," since they felt that their studies were undertaken for the good of their society. Similarly, they could have felt comfortable about Rule 3, which requires that the results should be such as to justify the experiment.

It would probably be too much to ask of Dr. Beecher or anyone else, at this stage of human affairs, that medical research be provided with a completely acceptable, honorable, and workable code of ethics.

ROBERT FARRIER, M.D.
Bethesda, Maryland

(Continued on page 36A)

BOOK REVIEWS

(Continued from page 35A)

The Physiological Basis of Diuretic Therapy

ROBERT F. PITTS, M.D., PH.D., 1959. Springfield, Ill.: Charles C Thomas. 295 pages. Illustrated. \$9.75.

This is a well-written book by an author who is both a physician and a professor of physiology. It is not concerned with the over-all management of the patient with edema and/or ascites. Rather, it is limited to a consideration of diuretic therapy and, more specifically, its physiologic aspects.

The content is divided into 2 parts. The first part concerns the volume, composition, and mechanism of homeostasis of body fluids and abnormalities in edema. This part is not so technical that a busy practicing physician is unable to read and readily understand these factors. The understanding is also aided by drawings and charts.

The second part has to do with the mechanism of action and the therapeutic use of diuretics. The various diuretics are placed in a functional classification as seen in a table. Then each of the diuretics in each functional classification is described in a following chapter. This discussion, for the most part, concerns the nature of the diuretic and its physiologic action on the kidney, intestine, and so on; any alterations in the composition of body fluids induced by the clinical use of diuretics; mechanism of diuresis; contraindications; and treatment of complications.

The final chapter deals with the diagnosis and treatment of hyponatremia and potassium deficiency, since

these conditions may occur in the course of therapy with any effective diuretic agent.

I believe I can recommend this book to the medical student, resident, or internist who would like to refresh his memory concerning the physiologic basis of diuretic therapy.

ROBERT S. WOODWARD, M.D.
Jamestown, North Dakota

That the Patient May Know

HARRY F. DOWLING, M.D., and TOM JONES, B.F.A., 1959. Philadelphia: W. B. Saunders Co. 139 pages. Illustrated. \$7.50.

The over-all impression of this book is that it is an excellent manual for use by the physician in counseling patients.

The section on nutrition, metabolism, and growth is especially well done and covers a subject which is usually most difficult to satisfactorily explain to patients.

In section 3, "The Respiratory Tract," I would like to have seen illustrations concerning pneumonia and carcinoma. The drawing of the x-ray of the normal chest was not too effective, and I believe a chest film would be a better visual aid.

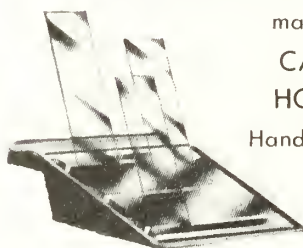
It was felt that some material on the action of anticoagulants and the employment of such agents in cardiovascular disease should have been included in either the section on the cardiovascular system or the section on blood.

(Continued on page 38A)

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BOOK REVIEWS

(Continued from page 36A)

Also, something on toxemia of pregnancy might be helpful, though some of this subject could be covered by reference to the nutrition section.

In summary, it is felt that the book has merit and should be of considerable value to the busy practitioner who must give a maximum of information to his patients in a minimum of time. I can see little of value in the book to medical students.

E. N. AKERS, M.D.
Denver

Essential Principles of Pathology

JOHN W. LANDELS, M.A., M.B., 1959. Philadelphia: J. B. Lippincott Co. 278 pages. \$5.00.

The purpose of this book, as stated in the introduction, is to give a general picture of pathology in relation to clinical medicine. It is not intended to cover morbid anatomy in detail.

The book does not present enough basic material on pathology to serve as a textbook for students of that subject. Nevertheless, it serves well as an auxiliary text and could very well be used as a preview to more complete works on basic pathology.

The section on inflammation is very good. The photographic plates are excellent, and the accompanying explanatory notes add to the understanding of them. The section on tumors appears to be somewhat too brief.

The print is a bit on the small size, but otherwise the book is well done; it has an adequate index as well as useful tables.

The medical student would find the book well worthwhile, especially at the beginning of his clinical course.

REUBEN F. ERICKSON, M.D.
Minneapolis

Atlas of Roentgenographic Positions

VINIA MERRILL, 1959. St. Louis: C. V. Mosby Co. 663 pages. Illustrated. \$32.50.

Since its initial publication in 1949, the two-volume "Atlas of Roentgenographic Positions" has served as a valuable reference text in departments of radiology. The book is concise and more nearly complete than any other similar volume available. The material is presented in an attractive fashion, and the drawings and roentgenogram reproductions are excellent.

In the preface to the second edition, the author states that a "reasonably complete reference work of roentgenographic positions should be as permanent as a standard dictionary—that it should not, at this stage of development, need frequent revision." The format of the second edition is identical to that of the first. The vast majority of the roentgenogram reproductions and photographs are identical. There have been only a small number of changes made in the reproduced radiographs. Several positions have been added to the book, many of these being from the old literature. A review of the bibliography fails to reveal a reference dated later than 1948, which is a year before the publication of the first edition.

In the initial portion of the first volume, there is a section on preliminary steps in roentgenography which includes some general and specific instructions regarding the handling of patients in the x-ray department and various other areas where radiographs may be made. In this present age of awareness of the undesirability of excess exposure to radiation, a section on radiation

protection for the patient, the technician, and the orderly or other individual frequently required to hold the patient would be a desirable addition. Instructions about limiting field size and gonadal protection would also be in order.

As was previously stated, these volumes, due to their excellence and completeness, have become a standard reference work for radiographic positions in departments throughout the country. However, after a careful comparison of the first and second editions, it is the opinion of this reviewer that there is not sufficient difference between the two editions to warrant the acquisition of the new volumes when the first are available for reference.

RICHARD H. GREENSPAN, M.D.
New Haven, Connecticut

BOOKS FOR YOUNG PATIENTS

Dear Little Mumps Child

Peter Gets the Chickenpox

MARGUERITE RUSH LERNER, M.D., 1959. Minneapolis: Medical Books for Children. Illustrated. \$2.75 each.

Dr. Lerner's colorful books, intended to explain common childhood diseases to the child on his own level, appear to be just the thing that the pediatrician and parent have been waiting for. Most children want to know *why*; many are frightened by the symptoms that attack them unexpectedly. In creating simple stories around her explanations, Dr. Lerner has made it possible for the child to learn about his illness without being involved in a technical description far above his head or insulted by a simple dismissal of the subject on the grounds that he "wouldn't understand." Delightfully illustrated by George Overlie, the books should be a source of positive guidance to parents as well as their sick offspring. What parent and child would not enjoy, for example, the hero Peter's description of his ailment: "Chickenpox is very itchy. It makes me feel all scratchy-scratchy." For the benefit of parents, each book contains a brief description of symptoms, etiology, incubation period, incidence, and duration of the subject illness, while the child is happily reminded in the end that, "Dogs catch cats. Cats catch mice. Chickenpox catches you once, not twice!"

Doctors' Tools

MARGUERITE RUSH LERNER, M.D., 1959. Minneapolis: Medical Books for Children. 16 pages. Illustrated. \$1.00.

The author's understanding of the fears and feelings of children is made clear by the presentation of this booklet designed to familiarize the child with the apparatus with which he will come in contact during a visit to the doctor's office or the hospital. Illustrations by George Overlie show everything from a scale to a sphygmomanometer, with more pictures telling the story of how each is used. Technical names may be hard for the very young child, but pictures help to make the doctor's tools friendly and well-known objects. The text could be improved by the addition of simple explanations in language easy for a child to understand; however, the last pages are concerned with explanations in the adult layman's terms, which can quickly be translated by a parent in answer to questions. The book is recommended to pediatricians and parents as an aid to greater cooperation of the child through a lessening of his natural distrust of the unfamiliar.

NANCY S. HAWKE
Minneapolis

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News Briefs . . .

North Dakota

DR. DUANE W. NAGLE, who recently headed the department of otolaryngology at the Veterans Hospital in Minneapolis, has joined the staff of the Fargo Clinic. He will work with Dr. Neill F. Goltz in the ear, nose, and throat department. A native of North Dakota, Dr. Nagle received his education at North Dakota Agricultural College, the University of North Dakota, and the University of Illinois. He has practiced medicine at Enderlin and was formerly a resident on otolaryngology at the University of Minnesota.

• • • •

DR. ELEANOR CRIM of Fargo has returned to India as superintendent of a mission hospital in Ambur. She will return to her Fargo duties after an eight-month stay. Dr. Crim first practiced in India from 1929 to 1940 and has been back there on a visit.

• • • •

DR. JOHN R. GOFF, Fargo ophthalmologist, has joined the Fargo Clinic. A native of St. Paul, Dr. Goff attended the University of Minnesota, North Dakota Agricultural Col-

lege, North Dakota University, and the University of Rochester, New York. He is president of the North Dakota Academy of Ophthalmology and Otolaryngology.

• • • •

DR. ZINAIDA WEINGARTEN, a graduate of the University of Vienna who came to the United States three years ago, is a new member of the medical staff at the state hospital in Jamestown. For the last two years, she has been a resident physician at the state hospital in Weston, West Virginia, and before that held the same position at the Delaware State Hospital in Farnhurst.

• • • •

DR. WESLEY E. LEVI of Bismarck has been certified a diplomate of the American Board of Radiology. A 1945 graduate of Temple University in Philadelphia, Dr. Levi served in the United States Navy for three and a half years and then practiced medicine in Omaha and Beulah. He is now radiologist at the Quain and Ramstad Clinic and on the staff of Bismarck and St. Alexius hospitals.

(Continued on page 42A)

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FOREWORD

CURRENT TRENDS IN TUBERCULOSIS CONTROL

THE ARTICLES RELATING to tuberculosis in this issue are consistent with a trend in changing emphasis in tuberculosis control which is world-wide in scope. This trend stems from the increasing evidence that isoniazid has proved to be so effective in the usual case of tuberculosis, when given in appropriate dosage and for a sufficiently long period of time and preferably taken in suitable combination with other drugs, that it can be used now in the community public health control program to hasten the day when tuberculosis will no longer be a public health problem.

The major recommendation of the Arden House Conference on Tuberculosis, held last fall and jointly sponsored by the United States Public Health Service and the National Tuberculosis Association, was to this effect and urged mobilization "of all resources for a widespread application of the scientifically demonstrated and medically accepted procedures of adequate therapy." It urged, as one of several corollary recommendations, the "assumption by state and local public health authorities of their responsibility for ensuring adequate treatment and rehabilitation of all patients with tuberculosis" (*National Tuberculosis Bulletin*, February 1960).

The Expert Committee on Tuberculosis of the World Health Organization, which met in Geneva last fall, stressed that "largely because of the introduction of effective antituberculosis drugs," it is now "possible to extend the program to countries which have not previously been able to launch an effective attack against tuberculosis." It stressed that tuberculosis control "should receive priority and emphasis both by the World Health Organization and by governments." The eventual eradication of tuberculosis, even though "this cannot be achieved in all countries in the near future," was recognized as a realistic objective (to be published by the World Health Organization).

Both reports stress the importance of evaluating the tuberculosis problem in the area under consideration; determining groups needing special emphasis;

conducting adequate case-finding programs; ensuring adequate chemotherapy of the patients discovered, preferably with an initial period of hospitalization whenever possible; and, finally, follow-up until the drug or drugs have been administered for the period indicated, usually about two years. Both documents also recommend chemotherapy of young children reacting to the tuberculin skin test, even in the absence of any demonstrable chest pathology, and note with interest current research which may extend still further the use of drugs in so-called "secondary chemoprophylaxis." In both documents, the increasing value of the tuberculin test is stressed from the standpoints of diagnosis, treatment, epidemiology, and public health administration.

Finally, there is a third recent document which is consonant with this trend. This is a resolution presented by the delegation of the National Tuberculosis Association to the Council of the International Union Against Tuberculosis at its fifteenth International Conference on Tuberculosis, which was held in Istanbul, Turkey, last September. This presented the reasons why it now seems realistic to envisage ultimate world-wide elimination of tuberculosis as a public health problem and called upon the World Health Organization to give such a target top priority. This was passed by the Council of the Union and was presented to the Executive Board of WHO at its meeting the last of January and first of February in Geneva by representatives of the Union. It received a sympathetic response on the part of the Executive Board, which directed that it be called to the attention of the thirteenth World Health Assembly, to be held in Geneva in May.

All of these efforts should help intensify efforts toward the elimination of tuberculosis in countries which have already made progress and stimulate effective programs in those countries not so fortunate, with the aim, no longer considered visionary, of ultimate world-wide eradication of tuberculosis.

JAMES E. PERKINS, M.D., *Managing Director*
National Tuberculosis Association

"It Is Not Best to Swap Horses While Crossing the River"

J. ARTHUR MYERS, M.D.

Minneapolis

MANY CITIZENS of Minnesota, including the bulk of professional health workers, may have forgotten or may never have known of the effort and sacrifices of great leaders in the last decade of the nineteenth century and the first two decades of the twentieth century to establish one of the best sanatorium systems in the world.

Apparently, the idea of providing a public sanatorium in Minnesota was first broached by Dr. L. A. Nippert in 1892. The next year, Dr. H. Longstreet Taylor moved to Minnesota and began to work unceasingly to establish procedures leading toward the eradication of tuberculosis. He was a strong supporter of the idea of providing sanatoriums where persons with contagious tuberculosis could be isolated. His article in the *Northwestern Lancet* in 1893 outlined the problem and discussed methods of solution. He and his disciples led the campaign throughout Minnesota for fourteen years before even a small part of their vision was realized.

A commission was authorized to investigate the subject and to select a site if it was found advisable for the state to erect a sanatorium. The report of this commission, which can be read with great profit today, appeared in the February 1903 issue of the *St. Paul Medical Journal*. The commission not only strongly recommended building a sanatorium but also selected a site near Walker. The institution opened its doors to 55 patients in December 1907, two years after Dr. Taylor had established a private sanatorium at Pokegama, near Pine City.

Since that small beginning in 1907, more than 13,000 patients have been admitted to the institution near Walker. The facilities of the organization to date, as presented by Dr. Williams in this issue of *THE JOURNAL-LANCET*, illustrate the fine provisions that have been made throughout the years to keep the sanatorium in the front line of institutions of its kind. As time passed, 14 other sanatoriums were established throughout the state by single or groups of counties. All were completed by 1919. By 1949, these institu-

tions, including the state sanatorium, had approximately 53,000 admissions. During the same time, 11,264 persons died in these sanatoriums. When one considers the uncountable numbers of tubercle bacilli that were corralled in sanatoriums, the hundreds of thousands of people who were protected against invasions from them because contagious cases were removed from their communities, and the many persons who recovered, it becomes obvious that sanatoriums played the dominant role in bringing about the phenomenal decrease in the destructiveness of the disease.

In 1907, when 55 beds were made available in the state sanatorium, 2,039 persons were reported to have died from tuberculosis in Minnesota. In 1911, one year before the first county sanatorium was opened at Nopeming near Duluth, 2,522 died, a death rate of 119.7 per 100,000. As sanatorium beds increased, the mortality rates decreased so, by 1958, only 105 persons died, a mortality rate of 3.2 per 100,000.

With the decrease in mortality, there was a corresponding decrease in morbidity. For example, just after all of the sanatoriums were built in 1919, there were 5,997 reported cases, whereas, in 1958, there were only 1,151 such cases. About 1919, it was estimated and actually determined in 1926 by testing that 70 per cent of eighth-grade children in Minneapolis had been invaded by tubercle bacilli, whereas, by 1954, this percentage was about 8.

ERADICATION OF TUBERCULOSIS—THE GOAL

The founders of sanatoriums and those numerous dedicated persons, including physicians, nurses, social workers, and other members of sanatorium personnel as well as the citizenry, who have so adequately supported them, had not merely control but eradication of tuberculosis as their goal.

In numerous activities of life, it has long been observed that many persons work enthusiastically and untiringly on health and other projects until the glamour is gone and the drudgery of

the final clean-up begins. The number willing to carry through to complete accomplishment is greatly limited, which is apparently why so many worthwhile projects are never completed.

A striking example of man's failure to eradicate a pest has to do with the English sparrow first brought to North America through the efforts of the directors of the Brooklyn Institute in New York in 1850. This is also an example of an error resulting from personal opinion, sentiment, and so forth rather than established facts. Instead of being helpful in controlling caterpillars and various insect pests, as opinion had it, this bird is 96 per cent vegetarian and therefore consumes large quantities of grain grown for domestic animals and people. After the first importation, it became such a fad to introduce these birds that, by the 1880's, they had been liberated in 33 of the states and Canada. Not only does this species consume huge quantities of grain but it fights desperately and crowds out many of the most beautiful native songbirds. In 1912, Chapman, curator of Ornithology in the American Museum of Natural History, said, "Its harsh, insistent, incessant chirp is now the dominant bird voice about our homes where we may never again hope to hear a chorus of native bird music unmarred by the discordant chatter of this alien."

When the English sparrow was found to be a pestiferous species, attempts were made to eradicate it, which began first in Michigan in 1887 and then extended to several other states. The method consisted of the state paying a bounty of \$.01 or \$.02 a head. A bounty of \$.02 apiece was authorized in Ohio where, as a small farm boy, I participated in the campaign. It was a simple matter to use a bright light and pick the birds off their roosting places or shoot with a shotgun into a large flock assembled in a small feeding area.

This was an effective procedure as long as a sizable number of birds could be bagged, but, when the total number was so decreased that a whole evening might net only one, a bounty of \$.02 apiece was too small. Moreover, the glamour was gone. As so few birds were being turned in for bounty, officials apparently developed a sense of security and the bounty was removed. Eradication might have been achieved if, when these birds became relatively scarce, the bounty had been increased to \$1 apiece, later to \$5, and, finally, to \$100 or more for the last birds in a community. Whatever the cost, it would have been insignificant when compared with the magnitude of the potential problem.

With the bounty removed and the glamour

gone, the few remaining sparrows multiplied rapidly. Soon English sparrows were as numerous as before the eradication effort began.

That a contagious disease can be eradicated from a nation was demonstrated in England with regard to leprosy. The contagiousness of this disease was recognized in Western Europe several centuries before Hansen discovered its cause in 1874. It had long been a common, chronic contagious disease in several parts of the world. In Western Europe alone, more than 20,000 leprosariums were provided for compulsory isolation of persons with this disease, 200 of which were in England. By the end of the sixteenth century, leprosy had been completely eradicated in England and was a rare disease in all Western Europe.

Probably it is because of the same psychology as manifested in the case of the English sparrow that no communicable disease of people, other than leprosy, has ever been eradicated. Despite the fact that we have known how to eradicate smallpox since 1796, more than 400,000 cases still occur in the world annually. With all that has been known about methods of eradicating diphtheria, approximately 1,200 cases and a few hundred deaths are reported in the United States annually.

We are experiencing an identical situation with the tubercle bacillus. When extensive sanatorium building got underway early in the century, enthusiasm ran high. Tuberculosis was then in the limelight. Members of numerous families were sick, and many people died. The problem was easily visualized and efforts to solve it were supported. With ever-increasing success of the tuberculosis eradication movement, as manifested by decreases in mortality, morbidity, and infection attack rates, one would expect that the entire citizenry, especially those who worked so hard to attain success, would take unlimited pride in their accomplishments to date and would cooperate to the nth degree in traveling the remaining distance to the final eradication of tuberculosis. However, with the disease markedly reduced and the glamour gone, complacency has reached high proportions among the citizenry, some tuberculosis organizations, and medical associations, despite the fact that there is evidence to indicate that tubercle bacilli have taken refuge in the bodies of approximately 50 million people in the United States (750,000 in Minnesota), of whom 60,000 to 80,000 become clinical cases and approximately 12,000 die each year. Although the number is far smaller than it was a few decades ago, it is still staggering to know that a new clinical case of tuberculosis is

reported every six minutes in the United States and that a person dies from the disease every forty-five minutes.

PREDICTIONS AND OVERENTHUSIASM PROMOTE COMPLACENCY

Numerous predictions have been made concerning the length of time that would be required to eradicate tuberculosis. For example, about 1920, a widely known tuberculosis worker said that if the desired number of sanatorium beds were provided promptly, tuberculosis would be defeated in twenty years. Another said that if those working in tuberculosis could have a grant of \$2 million, they would finish the job in ten to twenty years.

In 1940, such progress was being made that a well-known person predicted that the problem would be solved by 1960. Proponents of mass x-ray surveys promised rapid attainment of the tuberculosis eradication goal. Only a few years ago, a widely known physiologist said that, if a certain "immunizing agent" were generally accepted and used, sanatoriums would soon be obsolete. In 1949, with reference to the tuberculosis problem in Japan, it was said that an "immunization" program would prevent most of the cases of clinical tuberculosis from occurring in the future. In 1953, the statement was made that tuberculosis could be eliminated as a major social hazard within the next ten or fifteen years by the use of chemotherapy and resectional surgery. Recently, a prediction was made that in three years there will be only 75 cases of tuberculosis in Hennepin County, Minnesota.

Obviously, such predictions were not made by students of tuberculosis, and no truly informed student of the disease today would predict that the tubercle bacillus will be eradicated in the next one hundred years. Nevertheless, these predictions were made without an understanding of the disease, and they have had a detrimental influence on the eradication campaign. They have led the citizenry to believe that the solution of the problem is simple and the end of the disease is near. This has dampened enthusiasm for the long, hard path necessary to attain eradication.

Sanatoriums were built with the express purpose of reducing tuberculosis from generation to generation. Therefore, it was clearly anticipated that, as time passed, fewer people would be invaded by tubercle bacilli, with a resulting decrease in morbidity and mortality. For many years, most sanatoriums had many more applications than available beds. A great many people ill from tuberculosis remained in their homes

for a long period before sanatorium beds were available. However, as the effectiveness of the control of the disease in cattle and of sanatoriums in reducing infections in the general population reached a generally noticeable level, sanatorium waiting lists began to decrease. The time came when all applicants could be admitted promptly; then a few vacant beds began to appear, and their number gradually increased. To the student of tuberculosis, this was only an indication that success was being achieved; it did not justify the thought that the sanatorium was soon to be obsolete.

About 1947, mortality began to decrease more precipitously than morbidity. This change apparently was largely due to suppressive drugs and resectional surgery, which at least postponed death for many persons. Unfortunately, no anti-tuberculosis drug now available, or any combination of drugs, can cure tuberculosis. They are not germicidal. Moreover, resectional surgery applies only to lesions which are offending at the moment, not to the numerous other lesions in the individual's body which have not attained demonstrable clinical proportions. Therefore, present antituberculosis drugs and resectional surgery do not constitute a panacea for tuberculosis. Nevertheless, these agents and early diagnosis have reduced the average period of hospitalization to approximately one half the time required ten years ago. Thus, the capacity of sanatoriums for patient admissions has doubled.

Moreover, publicity pertaining to drugs and resectional surgery has convinced many physicians and a large number of citizens that these two procedures will solve all problems without hospitalization, except for a brief period in a general hospital while surgery is being performed. This is well emphasized in Dr. Williams' paper in this issue of *THE JOURNAL-LANCET*.

When some sanatoriums were closed because they did not have complete laboratory equipment to study resistance of tubercle bacilli to drugs or a first-class department of surgery, despite the fact that they had long been affiliated with excellent laboratories and other institutions where their patients had all the advantages of such procedures, and when sanatoriums were abandoned and reopened for admission of such groups as elderly individuals, complacency was greatly increased in the public mind, even among professional health workers not closely associated with the tuberculosis problem.

Complacency has extended to and is seriously hampering the local and national tuberculosis eradication program among the 97 million cattle of this country. This program has resulted in

accomplishments which have been designated man's greatest victory over tuberculosis. It is an exceedingly important part of the eradication program in people. However, in the last few years in at least 2 states, complacency resulted in decreased activity in the tuberculosis eradication campaign to such an extent that tuberculosis outbreaks began to occur to an alarming degree, and thus it became necessary to appropriate much larger sums for the program than would have been necessary if the original procedures had been continued.

CLOSING SANATORIUMS DISASTROUS TO ERADICATION PROGRAM

To close a sanatorium and to disperse its staff is to leave a job less than half done and to unleash the tubercle bacilli in the district it serves to disseminate from person to person until tuberculosis is again as widespread as it was before sanatoriums were established. One wonders if the closing of sanatoriums and other acts resulting in complacency in Minnesota may be in part responsible for the *increase in mortality of 20.95 per cent in 1959 over 1958*. In fact, more people died in 1959 from tuberculosis than during any year since 1955. That year, 129 died, followed by 115, 104, 105, and 128 in 1956, 1957, 1958, and 1959, respectively.

A disease which, since prehistoric days on the plains of the Ganges, has been so destructive as to be the first cause of incapacity and death and which still takes more lives than all other communicable diseases combined, today constitutes a special problem so large that it cannot be combined with any other disease problem if tuberculosis is to be eradicated. Tuberculosis is contagious and is notoriously a relapsing condition. Nothing has yet become available that destroys all tubercle bacilli in a human body; therefore, in the strict sense of the word, it is not a curable disease. Extirpation of all demonstrable lesions at the moment will not cure tuberculosis. In persons so treated, numerous other lesions, which are too small or so located that they cannot be demonstrated, are left in various organs. All of these are capable of resulting in clinical disease any time during the remainder of the individual's life. In many persons, tuberculosis is a lifetime disease because the tubercle bacilli, which invade the body during infancy and subsequently perpetuate themselves throughout the remainder of the individual's life, constantly threaten health and life itself. Although present treatment has greatly reduced the average length of time required to bring about an arrest of the disease, such arrest still requires often up to a year or

longer. The arrest of a lesion by no means guarantees that it will not later reactivate or that new lesions will not appear. For example, from the date of opening in 1916 to the present time, 24 per cent of admissions to Glen Lake Sanatorium have been readmissions.

QUALIFICATIONS OF SANATORIUM PERSONNEL

Sanatorium staffs, including physicians, nurses, and social workers, must have an understanding of the natural history of tuberculosis in a patient's body. They must understand the psychology of the chronically ill individual, and they must know the epidemiology of tuberculosis, the earliest and best diagnostic procedures, the best therapeutic measures and when to administer them, and preventive measures of proved efficacy. The sanatorium staff must know how to transmit information effectively to the patient's family, employer, and all others concerned, so that the patient will be aided in living with the disease for the remainder of his life. Graduation from a professional school or years of experience in other aspects of health work alone do not qualify physicians, nurses, or social workers to successfully work with tuberculous persons.

Truly successful sanatorium staff workers have a personal interest in tuberculosis. A considerable number of them come from sickbeds, through long periods of graduated exercise, to their official positions. Many have taken special postgraduate instruction and fine qualifications have been acquired through actual experience in dealing with groups of tuberculous patients. No amount of study can remotely approach the value of experience, but when such study and experience are combined, the truly qualified worker evolves. Such workers have staffed the sanatoriums in the upper Midwest and, in fact, throughout most of this country from the beginning of the sanatorium movement. Indeed, the first superintendent and medical director of the Minnesota State Sanatorium, Dr. Walter J. Markley, had the experience of having been the first superintendent of the first state sanatorium in the nation at Rutland, Massachusetts, before accepting the Minnesota post. Such persons have been and still are dedicated workers, devoting their lives to the eradication of tuberculosis. To close the institutions in which they work and make no provision for their continued services in the areas served leaves voids which cannot be filled.

CONVERSION TO DUAL PURPOSE INSTITUTIONS

To avoid disaster in the tuberculosis situation by closing sanatoriums and dispersing their

staffs, in some places, including the Province of Ontario and the State of Wisconsin, the provision was made whereby vacant beds became available to persons with other chronic, nontuberculous conditions. For example, at Fort William Sanatorium, Ontario, as the number of patients gradually decreased and the required length of hospitalization diminished, this plan was adopted without interrupting the work of the tuberculosis staff and with the understanding that tuberculous patients would be admitted promptly. This permits the staff to do whatever is necessary to care for the inpatients and increase its work in the field.

It now appears that Wisconsin will continue to operate its present 17 sanatoriums. The combined efforts of the persons best informed on all aspects of tuberculosis have resulted in the development of a highly satisfactory solution to the vacant bed problem, and already 8 sanatoriums have been converted to dual purpose institutions. Some operate as sanatorium-homes, others as sanatorium-hospital institutions. In each place, the highly qualified tuberculosis staff accepts all applications for tuberculous patients and provides the best known treatment for them.

In Wisconsin, the total number of patients admitted to the 17 sanatoriums was 3,004 in 1957 and 3,100 in 1958. These patients came from the large backlog of individuals harboring tubercle bacilli, most of whom probably were invaded in childhood. There, as everywhere else, the future number of new clinical cases from year to year will depend upon the backlog of infected people, that is, those who react to the tuberculin test. There is little hope that the part of the backlog composed of persons in the upper age brackets will decrease, except as they are removed by death. Only a small part of the backlog exists in children and young people, because they have been so well protected against infection with tubercle bacilli. As they replace the older people, there should be little clinical tuberculosis among them. Therefore, the future of tuberculosis depends upon the number of persons now infected and those whom they may infect. Each sanatorium staff has a tremendous role to play in managing this situation.

The only way a sanatorium district can determine the magnitude of its tuberculosis problem is by testing its entire population with tuberculin. Where this has been done on a county-wide basis in rural areas, 50 per cent or more of those in the upper age brackets react to tuberculin, whereas usually no more than 2 or 3 per cent of grade school children react. All reactors should be found, as they and those whom they

may infect constitute future clinical cases. It is a large task for a sanatorium staff to learn where all the tubercle bacilli reside in the areas served, but it is the only method of learning whom to examine promptly and periodically thereafter.

Periodic retesting of all persons who do not react to the tuberculin test is also a considerable task, but it must be done if eradication is desired because the converters constitute an exceedingly important group. Their sources of infection should be promptly sought and they themselves must come under periodic observation. While physicians in general practice and nurses residing in the sanatorium district should regularly participate in tuberculin testing, such programs should be organized and directed by highly qualified sanatorium staffs in order to insure continuity and thoroughness. Periodic examination of tuberculin reactors enables one to find those in whom clinical lesions are destined to evolve long before such lesions become contagious or cause illness and when they are most treatable.

Thus, in Wisconsin, the tuberculosis work is not being interrupted by closing institutions and dispersing staffs. Those institutions are continuing to serve as in the past. The scene has changed from the day when a preponderance of patients had far advanced, hopeless disease and large numbers of persons were on the waiting list to the present when all persons in whom clinical tuberculosis develops can be promptly admitted and given the best known treatment. However, their number is small enough to allow staff members more time to reexamine former patients periodically, to observe carefully those discharged but still on drug treatment, and to comb the area served to find the location of all tubercle bacilli and act accordingly.

In the 17 Wisconsin sanatoriums, outpatient treatments numbered 34,793 in 1955 and 32,961 in 1957. Thus, approximately 10 times as many outpatient as inpatient treatments are being given.

TIME AND PATIENCE OF THE ESSENCE

Failure to recognize how much time and patience are necessary to eradicate tuberculosis can result in serious interruption of the eradication program. One must not forget that the disease is probably as old or older than the human race itself and that the germs have now taken refuge in the bodies of more than half of the 2,700,000,000 people of the world. Many of these germs are in lesions which have lost their blood supply; thus, there is little hope of ever destroying them by any drug or removing them

surgically. One must patiently wait and watch persons harboring these germs and act when necessary to control whatever clinical lesions may evolve to prevent them from becoming contagious. The patience required becomes obvious if it is realized that an infant whose body becomes invaded with tubercle bacilli today may need to be kept under surveillance for the next seventy or more years. To become impatient and not provide for such work would be to lose all that has been accomplished to date.

A fine example has been set by the veterinary profession and its allies, both from the standpoint of patience and action. When a nationwide tuberculosis eradication campaign for cattle was established in 1917, many persons thought a few years would suffice to attain the goal. It seemed a simple matter to find all animals harboring tubercle bacilli with the tuberculin test and to eliminate them. However, when Dr. J. A. Kiernan, chief of the Tuberculosis Eradication Division of the United States Bureau of Animal Industry, was asked what time would be required, he replied, "There absolutely are no grounds upon which a reasonable estimate can be made of the number of years it will take to eradicate this disease. All one can do is make a guess as to the time, and it is my belief that if the nation succeeds in eradicating tuberculosis in fifty years, it will be one of the greatest heritages our successors will have handed down to them." Then and since, veterinarians have realized that as long as there is one tuberculin reactor animal, a tuberculosis problem among cattle remains, and therefore all animals must be tested periodically. Forty-three years have passed since Dr. Kiernan's statement was made and, while the disease has not been eradicated from cattle, it has been brought to such low ebb that cattle owners are saved \$150,000,000 per year.

In Minnesota, the State Livestock Sanitary Board has had a clear vision of the importance of periodic testing of the cattle population. Under the direction of Dr. Charles E. Cotton from 1919 to 1942 and, for the past eighteen years, Dr. Ralph L. West, the disease has been so decreased in cattle that it now is necessary to test 100,000 to find a single infected animal. However, even with so few infected animals, periodic testing often reveals small outbreaks which, if they had not been found by the tuberculin test, could have resulted in a situation as serious as when the program began.

This patient effort on the part of veterinarians and their allies has also played a large role in reducing the incidence of tuberculosis in people

which was contracted from animals. In the early days of the Gillette State Hospital for Crippled Children in St. Paul, approximately 50 per cent of admissions had tuberculosis of the bones and joints. No such case has been admitted during the past year. There is reason to believe that pasteurization and the eradication campaign among animals reduced cases of tuberculosis of the skin and peripheral lymph nodes in people 50 per cent or more; of the skeletal system and genitourinary tract, 20 per cent; acute fatal forms, including meningitis and generalized military disease, 25 per cent; and pulmonary tuberculosis, 1 per cent or more. Hence, continuation of periodic testing of cattle is an essential part of the tuberculosis eradication program, not only in animals but also in people, and therefore should be supported unrelentingly.

TOO MUCH EMPHASIS ON VACANT BEDS

It is unfortunate that in recent years so much emphasis has been placed upon vacant sanatorium beds, at times almost reaching fanatic proportions, particularly since this item is such a small fraction of the tuberculosis eradication program. To become "economy minded" about vacant beds in sanatoriums is to demonstrate a lack of knowledge concerning the value of these institutions. If the lock were turned on every door of every room of every sanatorium in the Upper Midwest, or in the entire nation for that matter, and were never unlocked, they would still represent one of the best investments their communities ever made. They have paid for themselves over and over. However, there is no institution where the lock should have been or, for a long time, should be turned. There is no sanatorium area that has become destitute of clinical cases and large numbers of infected persons. Therefore, by continuing to operate as many of the institutions as are necessary for whatever number of clinical cases there may be and to comb the areas served for the last tubercle bacillus, the institutions will continue to pay for themselves over and over.

To use vacant beds in sanatoriums for persons with nontuberculous conditions, as approved by the medical profession and others concerned, is a good arrangement as long as adequate space within the institution is provided for clinical cases of tuberculosis and the staff is maintained to carry on to eradication of the tubercle bacillus.

Consolidating sanatoriums with the resultant elimination of one or more staffs is contrary to the thinking of the founders of these institutions. After careful consideration, they decided that, except in sparsely populated states, it was better

to have a number of small institutions throughout the state than one large sanatorium to serve the entire state. Their reasoning is as good today as it was then.

The thought of treating tuberculous patients in *general hospitals* was given most careful consideration prior to and during the days when sanatoriums were first being built, but the idea in most places was abandoned in favor of special hospitals for the tuberculous. Admission of tuberculous persons to general hospitals was advocated in the 1920's and 1930's largely because it enabled many contagious cases to be hospitalized and isolated earlier than was otherwise possible on account of long sanatorium waiting lists.

For prolonged treatment, tuberculous patients require more highly specialized staffs than usually exist in general hospitals. Moreover, the general hospital environment is not satisfactory. A special tuberculosis and chest disease service has existed at the Minneapolis General Hospital since 1938, but it has not been regarded as a treatment center. Its function has been for prompt admission of emergency cases, differential diagnosis, and isolation of contagious cases until they can be admitted to sanatoriums.

The outstanding example of a general hospital operating an excellent tuberculosis service was conceived by Dr. H. Longstreet Taylor, who recommended that the Ramsey County Pavilion for tuberculosis be constructed on the city and county hospital grounds as a separate building. It has been under the administration of the Ancker Hospital but has been operated by a highly specialized staff as a sanatorium.

AN UNPREDICTABLE DISEASE

Tuberculosis is an unpredictable disease. Development of its clinical forms depends upon such factors as the size of the backlog of infected persons and the number being infected annually. A number of situations, such as unfavorable social conditions, could result in slight to marked increase in clinical cases. Thus, the fact that the mortality rate has been steadily decreasing for a decade is not a sure criterion that the same rate of decrease will continue. There is no mathematical formula or slide rule procedure to make such determinations. In Hennepin County, Minnesota, for example, there is good reason to believe that 25 per cent, or 205,750, of the 823,000 citizens are now harboring tubercle bacilli. Estimates of the number of such infected persons who will develop clinical tuberculosis range from 5 to 50 per cent. If the 5 per cent estimate is correct, 10,387 of the

205,750 presently infected persons will develop clinical tuberculosis, to say nothing of the number becoming infected annually. No one can know whether there will be 50 clinical cases by 1965 or half that number or twice as many. The item of greatest importance is that whatever the number, these individuals must receive the same expert sanatorium treatment that the 17,156 admissions to the Glen Lake Sanatorium have received since 1916 until the present time. Of the total number of admissions, 4,240, or 24 per cent, were readmissions, indicating the relapsing nature of tuberculosis.

COST

The nearer the eradication goal, the greater the cost per individual of finding where tubercle bacilli reside, but this is compensated manyfold by decrease in other costs through the reduced number of clinical cases. For example, in Minneapolis, as much time, effort, and funds were required to find 4 infected grade-school children per 100 tested in 1954 as to find 47 in 1926. However, seeking the source of infections and keeping the 4 per 100 under surveillance cost far less in 1954 and subsequently than did the same procedure for the 47 in 1926. This also applies to clinical cases which should have sanatorium management. The per diem cost of operating a sanatorium of 300 beds is definitely less when all are occupied than when the census is reduced to 50. Nevertheless, it is just as important to keep sanatorium beds in operation for 50 persons or less as it is for 300, and it will be equally as important to keep an adequate number of sanatorium beds available as the number of persons requiring such management approaches zero.

As the number of persons requiring sanatorium management decreases, the sanatorium's professional staff is able to devote more time to the finer details of the eradication program, namely, locating all tubercle bacilli among the citizens of the area served. This is time-consuming, meticulous work but is so indispensable that the sanatorium staff should not only be retained but should have additional members provided.

QUALIFIED PERSONNEL ESSENTIAL

Persons thoroughly informed and experienced in the various aspects of tuberculosis should continue to be in charge of all phases of the eradication program, including management of institutions. Uninformed hospital managers could take over sanatoriums and make annual "savings" of \$1,000 to \$1,000,000, depending on the size of the institution, by eliminating certain

services and activities which they do not understand. Abolishing these services, however, can be "penny-wise." Some of them are so important to the tuberculosis eradication movement that their exclusion could cost the area an unnecessary additional \$50,000 or \$50,000,000 in the next forty or fifty years. Tuberculosis is often a lifetime disease, and its chronic forms are slow and insidious in development. Elimination of services now that would permit wide dissemination of tubercle bacilli would be little felt in five or ten years but would strike terrific blows twenty-five or more years later.

Sanatorium boards and commissions must continue to consist of informed men and women who are devoted to the cause of tuberculosis eradication. They must see to it that only well qualified, devoted, and sincere persons are employed to operate institutions.

The accomplishments leading toward eradication of tuberculosis in the upper Midwest have been phenomenal. From the beginning, the work has been directed by students of tuberculosis, social workers, nurses, veterinarians, physicians, and many others who armed themselves with the best knowledge of the day and thereafter kept abreast of the latest developments wherever they occurred. The soundness of their program is evinced by accomplishments which, in some states of the upper Midwest, surpass those in any part of the world, except Iceland. They are far ahead of the tuberculosis timetable

schedule. But, because of such factors as the nature of the disease, the span of human life, and so forth, they are scarcely midway toward the goal of tuberculosis eradication. However, these workers have a clear vision of the goal, and they know the best course by which it can be reached. This group may be likened to those with expert knowledge and experience in various other activities of life. For example, few persons would care to board an airliner destined for such places as New Delhi or Moscow which was manned by a crew with only reading knowledge or hearsay of such an operation. The catastrophe that most likely would occur during the take-off or en route would be insignificant compared to that which would occur if a group of individuals armed only with personal opinion, hearsay, speculation, and theory were to take charge of the tuberculosis eradication program.

The solution to the tuberculosis problem, which pertains to health and the very lives of our citizenry, requires the wholehearted support of every citizen. After the fine accomplishments to date and work presently in progress leading directly toward the eradication goal, it would be a blight on this generation if any action were taken to halt progress.

Certainly, no citizen will permit swapping horses while crossing the river, and none will ever want it said that "what one generation built another destroyed."

TUBERCULOSIS AMONG currently examined Selective Service registrants has declined in incidence to somewhat less than one-third of that found within a comparable age group during World War II. The current rates are about 36 per 10,000 registrants for tuberculosis of all forms and about 32 per 10,000 registrants for respiratory tuberculosis. Among 177,000 registrants aged 18 to 26 years examined from January 1957 through March 1958 at Armed Forces examining stations, 20.3 per 10,000 had disqualifying tuberculosis of all forms, and 18 per 10,000 had respiratory tuberculosis. Incidence was higher among Negroes.

In addition, 12.4 per 10,000 registrants were disqualified by local Selective Service boards prior to AFES examination because of tuberculosis of all forms, and 10.7 per 10,000 were disqualified because of respiratory tuberculosis.

B. D. KARPINOS: Prevalence of tuberculosis among Selective Service registrants currently processed for military service. *Am. Rev. Respiratory Dis.* 80:795-805, 1959.

Let's Wipe Out Tuberculosis, Too!

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THE YEAR 1960 begins as a time of tremors and quakes, raids, and revolutions in almost all lines of human activity. This is as true in tuberculosis control as elsewhere. For months now, Minnesota tuberculosis workers have joined in increasingly earnest conferences, arguing, analyzing, exhibiting frustration, and throwing anxious questions but with growing determination to build a firmer tuberculosis control structure.

The tools of control are already at hand: (1) alert minds to suspect tuberculosis; (2) the tuberculin test to find infection; (3) chest x-ray films to find gross disease; (4) bacteriologic tests to prove the diagnosis; (5) examination and follow-up of known contacts of infectious cases to break spread of the disease; (6) chemotherapy, established in the sanatorium or hospital and supported by rest and surgery where indicated, for treatment of the active case; and (7) permanent follow-up of the significant case to maintain cure. The tools are well proven in usefulness. Questions today are not as to what tools to use but how better to apply them.

The problem may appear simple, but is it? In a recent strategy meeting of tuberculosis workers, we were reminded that one fourth to one third of Minnesota's population, perhaps 1,000,000 people, react positively to the Mantoux test and, therefore, are known to have been infected with the tubercle bacillus. This leaves something over 2,000,000 persons uninfected. For *ideal* control, all uninfected persons should be Mantoux-tested annually or periodically until all danger of infection is past. Will the more than 2,000,000 hold still for these injections, and who will pay for and do the tests? The realistic answer is obvious. We can well do pilot studies, that is, Mantoux surveys of selected groups or interested communities, but we cannot hog-tie the entire state even for a single coverage, let alone for periodic repeat surveys.

As for the close to 1,000,000 infected Minnesotans, each should *ideally* be intensively studied, both initially and periodically, according to his

own medical indications. Clinical follow-up should be for his lifetime. And who is to see that all of these examinations and procedures are thoroughly done, faithfully followed, and properly paid for? Again, realism gives us a harsh answer.

During the conference previously mentioned, it was noted that Hennepin County, with a population of over 500,000 and, therefore, presumably with a Mantoux-positive population of over 125,000, has a tuberculosis follow-up registry covering 3,000 individuals, or about 1 out of 40 of the persons with positive reactions. St. Louis County, carrying a population of over 200,000 and with at least 50,000 infected persons, has been proud of a registry of about 2,600 cases of tuberculosis plus a further 3,300 Mantoux positives, making a total registry of 5,900 out of about 50,000. Both counties are far short of ideal follow-up. They don't even approach it. Yet, if these records are poor, what county has a good record? Many counties have far less of a program than Hennepin and St. Louis. If Minnesota cannot be proud, what of our less fortunate sister states?

In view of costs and public resistance, we cannot expect to achieve the ideal and must stop somewhere within the limits of realism. Of concern to all of us is the fact that many significant cases, not merely of Mantoux positivity but of proved minimal, moderately advanced, or far advanced tuberculosis are also lost to follow-up. These are serious losses, for we know that some of these persons will suffer reactivation of disease and may lose life or health or may seriously infect other persons. Robert Ragsdale, executive secretary of the Hennepin County Tuberculosis and Health Association, has summarized the problem thus: "Not all of the cases are being found; of those found, certainly not all are receiving adequate medical care and supervision; of those receiving care, not all are accepting it wholeheartedly or staying until the problem is resolved."

In other words, our control work is incomplete, uneven, and spotty. It frequently overlooks large geographic, social, and medical areas. The incidence of tuberculosis in the state is

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being gradually reduced but far more slowly than the public realizes. Case finding, diagnosis, and follow-up activities must not be allowed to lag. As inpatient treatment periods are further gradually shortened, sanatorium populations decline and sanatoriums are closed on the basis of administrative "anemia." Outpatient facilities must be maintained and, in fact, be expected to incur increasing responsibility.

We should maintain all county and local control activities now available, but, in addition, marked strengthening and uniformity in control efforts throughout the state should be developed through a system of state or district epidemiologic supervisors armed with authority to investigate and control the careless, heedless, or

recalcitrant individual, family or community focus. Provision by the state of free care for the tuberculous patient would also be of help.

In summary, then, we must: (1) maintain and keep sharp the tuberculosis tools available; (2) maintain, broaden, and strengthen our state and local case registry systems; (3) close inpatient facilities with caution and only as they become medically inefficient; (4) maintain a vigorous outpatient facility in every district for promotion of case finding, supervision, and permanent follow-up; (5) provide free care for all state residents with active tuberculosis, and (6) establish and maintain a vigorous state epidemiologic control system with statutory authority adequate to eradicate tuberculosis.

SIMULTANEOUS INTRACUTANEOUS testing with standard mammalian tuberculin (PPD) and with antigens prepared from other types of mycobacteria may indicate whether tuberculin sensitivity is caused by infection with virulent bacilli or with cross-reacting organisms.

Tests with PPD and with an antigen made from the Battey mycobacterium were made using experimentally infected guinea pigs and patients in tuberculosis hospitals. In most cases, accurate differentiation of tuberculous infection and that with organisms of the Battey type was possible. The homologous antigen usually produces a larger area of induration than does the heterologous one.

Geographic variation is evident in frequency of cross reactions to tuberculin in man. In those areas in which cross reactions are common, the tuberculin test alone may not be relied upon for accurate diagnosis.

In relation to the decreasing incidence of tuberculosis, infections with cross-reacting mycobacteria are appearing with increased frequency. The Battey organism, a nonphotochromogenic acid-fast bacillus, appears to infect and sensitize many persons, although it rarely causes evident illness.

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Epidemiologic Aspect of Chemotherapy in Tuberculosis

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THE ADVENT OF CHEMOTHERAPY in tuberculosis about fourteen years ago brought about the most significant advance known in the effective treatment of this disease. Mortality rates declined rapidly, and patients with positive sputum achieved the goal of negative cultures in unbelievably short periods of time.

All this was and is most gratifying. Unfortunately, the indiscriminate use of chemotherapy may have had an adverse effect on the control and eventual eradication of tuberculosis. Since my endeavors have been directed almost entirely toward control of this disease, my interest is mainly in the epidemiologic aspect of the use of chemotherapy.

However, we will take time to mention those drugs used in the treatment of tuberculosis which have been fairly well-established. Three drugs now commonly used are (1) dihydrostreptomycin, an antibiotic which replaced streptomycin because it showed less toxicity; (2) para-aminosalicylic acid (PAS); and (3) isonicotinic acid hydrazid, or isoniazid (INH).

An adult of average weight is given 1 gm. of dihydrostreptomycin intramuscularly, usually in the gluteal region, daily for thirty days, then three times weekly. The length of treatment depends on many factors: progress of the patient, roentgenographic findings, and bacteriologic study and culture of sputum and gastric washings. One must bear in mind that a negative culture does not have quite the same meaning now as it did before the use of chemotherapy.

Toxicity to dihydrostreptomycin is usually manifested by an impairment of hearing, some tinnitus, and some vertigo. Streptomycin sulfate is sometimes preferred because it causes less deafness, but it may cause more vertigo.

As a rule, PAS is used in conjunction with

dihydrostreptomycin and is usually given by mouth. The daily dose necessary to ensure adequate plasma concentration is large and depends on the weight of the patient. At the present time, the dose for a 160 lb. adult is 12 gm. a day in 4 divided doses. If intolerance to PAS is manifested by gastric distress, flatulence, decreased appetite, and, possibly, nausea, dosage of the drug should be decreased or discontinued.

INH is used in conjunction with one or both of the drugs mentioned. It is administered by mouth, as it is rapidly absorbed through the alimentary tract. The dosage for a 160 lb. adult is 200 to 300 mg. daily. The drug induces practically no toxicity or hypersensitivity.

The combination of these 3 drugs in full dosage is used because it has been shown to be the best method to date of avoiding the development of drug-resistant tubercle bacilli. Other drugs used are (1) viomycin, to be used only for short periods under close supervision, often immediately preceding thoracic surgery; and (2) adrenal steroids, used in combination with other chemotherapeutic agents, which "have shown definite value in patients with miliary tuberculosis, tuberculous meningitis. . . . Its use in less severe forms cannot be routinely advocated."

There are several other drugs now being studied, especially for use in cases in which resistance to established therapy has developed. These should be used only where careful scrutiny and study of the patient is possible.

The discovery of these drugs makes it possible for the private physician to treat tuberculosis in the home much more readily than in the past, but is this a wise procedure? In certain instances, the answer is "Yes," but, in many cases, the answer is "No!" In all cases, certain important factors should be carefully considered:

1. Does the patient have positive sputum or positive gastric washings? If so, he is contagious.

2. What are the home conditions? Can the patient, besides receiving drug therapy, also get adequate rest, quiet and food? These are still

From the Riverside Sanatorium. Presented before the Minnesota Academy of General Practice, Minneapolis, September 22, 1959.

extremely important factors in the treatment of tuberculosis.

3. Will the patient follow the rules of living necessary for his own improvement and for the safety of his contacts?

4. Are there children in the home, or does the patient have grandchildren who may visit him? Are there children in the neighborhood who are in the habit of visiting frequently in the home?

The American Trudeau Society, which is the medical group of the National Tuberculosis Association, advises "that patients with tuberculosis should be hospitalized as soon as the need for treatment is established and remain in the hospital until the disease is no longer communicable and until suitable home care is available."

When a physician undertakes the care of a case of active tuberculosis, he assumes a double responsibility. The first involves a very careful study of the effects of the drugs used. Frequent gastric washings with guinea pig inoculations should be made to determine whether the treatment is adequate. If possible, systemic reactions to the drugs should be observed daily during the initial period of their use and frequently thereafter.

The second responsibility is that involving the epidemiology of the disease. To quote Dr. Herman Kleinman, head of the Division of Communicable Diseases, Minnesota State Board of Health, "The advent of chemotherapy does not excuse laxity in epidemiology." From a practical point of view, just what does this mean?

First, when a case of tuberculosis is diagnosed, every effort should be made to discover the infectious agent, whether of human or animal origin. In this endeavor, the State Board of Health stands ready to give advice and assistance. The public health nurse can be called on for help. Caution should be the key word in this search.

Often, the apparently obvious source of infection is not the true one. Such was the case of a small rural school in central Minnesota. The teacher of this one-room school was discovered to have active, contagious tuberculosis during the summer vacation and was hospitalized. The following fall, when school reconvened, I was asked to administer a tuberculin skin test to the pupils of this school. Sixty-five per cent of these children reacted. It would have been quite easy to have attributed these reactions to the teacher of the previous year. However, on closer study, it became quite obvious that the reactors were in family groups. There were as many reactors among first graders who had never been taught by the tuberculous teacher as among the rest of the pupils. After careful investigation, it was

found that the mother of one of the reactors and her older daughter were suffering from active pulmonary tuberculosis. The teacher had resided in that home.

Second, every possible contact of the patient should receive the Mantoux test, and all reactors should have chest x-ray film examinations. These roentgenograms are to be repeated annually, unless the original findings show the need of more frequent examinations.

Third, a tuberculous patient should never be treated in a home where there are children. However, all children having any possible contact with such a patient should be tuberculin tested every six months if found to be negative originally. Any child becoming a converter, that is, changing from a negative to a positive reactor to tuberculin, should be carefully studied and, according to certain authorities, treated with INH, even when all other findings are negative.

Perhaps the greatest danger we now face in regard to tuberculosis is one of overconfidence, even apathy. The decreased mortality rates, partly due to chemotherapy, tend to lull us to sleep. Morbidity rates are not as encouraging, but they certainly are not what they used to be twenty-five or thirty years ago. The upward trend in percentage of reactors in some of our high schools is disturbing. Incidents such as the much publicized Heron Lake episode cause only passing concern. We really need a renewed awareness on the part of all practicing physicians of the fact that tuberculosis is still a contagious disease and a stealthy one. One undetected contagious case may do untold damage. We have on record the case of a ninth-grade boy who infected 93 per cent of his classmates; 3 of those so infected later died of tuberculosis. This could happen again any time we let down our defenses.

In many parts of this state, tuberculin-test surveys of school pupils and personnel have been conducted for many years. In some sections, these surveys are made at definite and regular intervals. In other areas, they have been quite irregular. Well-established routine tuberculin tests of both pupils and personnel of our schools, with regular chest roentgenograms of those who are found to react to tuberculin and careful follow-up work to determine hitherto unknown sources of infections, are among the easiest methods of keeping track of tuberculosis in the community. Both the State Board of Health and the Minnesota Tuberculosis and Health Association are ready to give help in getting such surveys under way.

Establishing a safe school environment is not

sufficient. We must endeavor to establish as safe a community environment as possible. I suggest the following:

1. All hospitals should institute routine chest roentgenograms of patients on admission. This routine measure is practiced in a great many hospitals, but many more have not seen fit to use this definite protection. For a routine admission, an x-ray film is of value not only for diagnostic purposes but to protect the hospital staff.

2. The tuberculin test should be used routinely by the private physician in his office. This use was recommended by the Committee on Tuberculosis of the American College of Chest Physicians at their meeting in Atlantic City in June 1959.

Routine testing may avert the tragedy of an erroneous diagnosis. In a small town in southern Minnesota, a young woman was treated for three or four months as a case of unresolved virus pneumonia. She was discovered as a far-advanced case of pulmonary tuberculosis only when her roommate, who was a senior in high school, showed a marked reaction to the tuberculin test. This young high school girl had been a nonreactor during her previous school years. The school she attended offered the tuberculin test as a routine measure every two years. She had moved to town from a farm for her senior year in order to be gainfully occupied after school hours. Fortunately, the school believed in regular routine testing, even though the tuberculosis rate in that area is one of the lowest in the state.

3. For the safety of all, certain groups of people should be checked for tuberculosis regularly to protect the public they serve.

Besides school teachers, personnel, and school bus drivers, who are already being checked quite thoroughly, we would add food handlers, night club employees, employees of any public place where our teen agers are apt to congregate, bar-

bers, beauty parlor operators, and baby sitters to the list of those who should be regularly tested for tuberculosis.

This last group is most important. At the present time, most of our active tuberculosis is found in the section of the population over 50 or 60 years of age. Often mothers prefer older women as baby sitters for very small babies and high school baby sitters for the more active 3- or 4-year-olds or older. In Heron Lake, the person discovered with active contagious tuberculosis had not only been connected with the schools as the superintendent's secretary but had also been a highly valued baby sitter. At least 2 of the children that she had cared for showed definite roentgenographic findings. Perhaps each baby sitter should be checked yearly and carry a card stating the date of the last check-up and the report of the findings.

4. When a community shows more cases of tuberculosis than surrounding similar areas, or shows a marked increase in the percentage of reactors in the school, community wide tuberculin surveys are suggested. These surveys are usually sponsored by the Minnesota Department of Health, the local Public Health Service, or the Minnesota Tuberculosis and Health Association. Christmas Seal funds are used to help defray the expenses of the survey.

We have strayed far from the restricted subject of chemotherapy in tuberculosis, but the goal of all work in this field is the control and eventual eradication of this disease. We have the necessary tools with which to reach this goal—the tuberculin test, the chest x-ray film, and other diagnostic laboratory procedures plus a systematic, unrelenting follow-up of reactors to tuberculin to discover the source of infection and provide adequate therapy. These tools, used with persistence and unrelenting ardor, give us the hope of effective control and possible eradication within the next two generations.

IN PREMATURE infants, extreme hyperkalemia associated with electrocardiographic alterations may accompany the respiratory distress syndrome. Prolonged conduction times were seen on serial electrocardiograms twelve to sixty hours after birth in 36 of 37 infants with chest retraction, expiratory grunting, and decreased entry of air by auscultation but no coexisting disease. Serum potassium concentrations were more than 7 mEq. per liter, and parenteral administration of glucose, insulin, and bicarbonate reversed hyperkalemia and conductivity changes.

R. USHER: Respiratory distress syndrome of prematurity. *Pediatrics* 24:562-576, 1959.

Minnesota State Sanatorium at the Half-Century Mark

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Ah-gwah-ching, Minnesota

THE MINNESOTA STATE SANATORIUM was and is dedicated to the care of the tuberculous patient. To us, care comprises isolation of the contagious and treatment of the acute and chronically ill, our goal being the discharge of the patient in a state of well-being in which he no longer represents a threat to those in his environment. Such a utopian program should result in control of this widely spread public health menace.

In the beginning, this hospital's function was limited by law to the admission and care of only the early, minimal, so-called incipient case of tuberculosis. To this original concept was added gradually, possibly illegally, the broader policy of admitting tuberculous patients in all forms and in all stages of the disease. So great has been the supply and demand that this hospital's population in 1939 reached a maximum of 436 patients crowded into space for 395. Evolution in a changing world of tuberculosis has, in recent years, permitted us to include a program of community case-finding procedures, diagnostic chest services for any physician or clinic in the state, and the all-important regular and periodic follow-up examinations of previously treated and apparently inactive cases annually, or at even lesser intervals, as indicated.

Since December 28, 1907, when the first patient was registered at the hospital, there have been in excess of 13,300 other patients admitted to date. Between these two hospital charts are written nearly all the clinical pages in the fifty-two-year saga of the Minnesota State Sanatorium—a tale of blood, sweat, and tears, sometimes

telling of hope renewed but all too often relating only of release from suffering in death. These more than 13,000 charts represent, I believe, a larger total of new admissions of patients than has been the experience of any similar tuberculosis sanatorium in Minnesota.

Tuberculosis is an infectious disease. Unlike some other infectious processes, it is also contagious. The tubercle bacillus has likely produced more ubiquitous and more manifest infection and disease, broken bodies, and death in mankind than has any other germ. Tuberculosis has been, is now, and will likely remain the one infectious, contagious disease most seriously threatening to man. We have in some ways improved our position in our fight against this scourge, but we have not yet won the fight. We have only regrouped our defenses and, in certain ways, have been able to make hard-gained, offensive thrusts at the enemy, but we have not vanquished it. The enemy is still there—always elusive, today's Jekyll and Hyde, resistant and obdurate. Even in this day of relative scientific success in our contest, the wily bug still stumps the experts and easily beguiles the less expert among us into assuming defenseless and wasteful attitudes and approaches in the long-term battle. All too often we, the profession, and they, the ill-advised tuberculous unfortunates, are throwing our punch while at the same time letting down our guard.

There may well be many reasons why an increasing number of once insufficiently numerous beds for the tuberculous are now empty in sanatoriums all over the nation. The obvious reasons have to do with the march of science—more specific drugs and subsequently safer surgery—which bring about a state of healing more promptly in most cases, thereby decreasing the duration of hospital stay. The less obvious reasons—the unwise failure of the profession in many instances to report this contagious disease to health authorities and, worse yet, to carry on its treatment at the business-as-usual level at home and not even temporarily under sanatorium

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Presented at the Minnesota State Sanatorium before Governor Orville Freeman; a group of state legislators; members of the State Mental Health Policy Committee; commissioner of Public Welfare, Morris Hurst; medical director of the Department of Public Welfare, Dr. Dale Cameron; president of the Minnesota State Medical Association, Dr. B. B. Souster; members of the press; and others.

isolation conditions—very realistically reduces, if not decimates, legitimate tuberculosis bed occupancy.

A study carried out in 37 widely spread urban and suburban areas in this country by our United States Public Health Service a few years ago revealed that as high as 45 per cent of the known, active cases of tuberculosis were not under isolation or treatment in recognized sanatoriums or hospitals. Only 10 per cent of these people were receiving any tuberculosis care or supervision of any kind. As many as 87 per cent of these ill-advised or recalcitrant tuberculous persons had a far-advanced, contagious form of the disease with cavities in the lungs capable of continued spread of infection to others. If this was true three or four years ago, what is the situation now? Better? Worse? The experienced among us view this with concern and alarm; some of the others must evidently not feel this concern.

From whence come the patients admitted to the Minnesota State Sanatorium? When the hospital opened its doors on December 28, 1907, patients were referred from every county in the state, there being no other tax-supported institution available. During its first year of operation, in excess of 60 per cent of admissions came from Hennepin County, 20 per cent from Ramsey County, and 10 per cent from St. Louis County. These counties later built their own hospitals or sanatoriums for tuberculous patients, some of which are still in operation but which suffer the chronic affliction of too few patients to fill their once crowded beds. Many of these empty beds would be occupied now by active tuberculosis cases if they were all being properly reported to the public health agency and if they were then referred by the physicians to the sanatoriums instead of being, in some cases, carelessly and unguidedly treated, or not treated, at home.

Within a period of about fifteen years after the state sanatorium began operation, more than a dozen other similar hospitals developed. Only one, Glen Lake Sanatorium, faced with nearly a third of the state's entire population, was larger than the state sanatorium, the true mother of them all. In spite of the creation of hundreds of more beds for tuberculous patients as a result of building additional sanatoriums, the state sanatorium stayed full and had a tragically long waiting list from more than 40 so-called unorganized counties. Not until 1939, nearly twenty years after the last of the approximately 15 tuberculosis hospitals had been in service, did the patient population at the state sanatorium reach

its peak. In March of that year, 436 patients were in residence in a hospital setting of about 395 beds. Beds were put up in corridors and visiting and reception rooms.

About 1935, a 120-bed building was erected by the federal government for the specific purpose of treating the veritable avalanche of sick and dying among the Indians of Minnesota. Other Indian patients were admitted from Wisconsin, the Dakotas, Iowa, and even Canada. In 1940, a tuberculosis chest surgical service was developed here under the consultative direction of Dr. T. J. Kinsella, thoracic surgeon of Minneapolis, and patients from the many smaller tuberculosis sanatoriums were referred to the state sanatorium for temporary surgical treatment. From 1955 to 1956, the Minnesota State Sanatorium medical staff furnished tuberculosis medical and surgical services, both by visiting consultation and by direct admission, for tuberculous inmates from the St. Cloud, Stillwater, Sauk Centre, and Shakopee penal institutions. Early in 1956, because of an increasing number of empty beds, a so-called "pilot study" was set up, and since that time, both tuberculous and nontuberculous mentally ill senile patients have been admitted for care as space and personnel became available. Our sanatorium is rated at 350 beds, but the introduction of the mental patient, with or without tuberculosis, has forced us to reduce our working bed capacity to a total of 307 beds to allow for full twenty-four hour living accommodations—eating, sleeping, and recreation—within the confines of any particular ward so converted to this use.

Any hospital is made up of a number of component departments. No department in a hospital is independent of the others. Without engineers and maintenance men, we would collapse physically because of lack of heat, electrical power, water supply, and sewage disposal. Without administrative business service, we would soon be without supplies of all kinds, and all functions relating to purchase, storage, disposal, requisition, and budget planning would be in chaos. Without clerical people in Medical Records, confusion resulting in catastrophic hodgepodge would result in a few days. The same could be said of each department, each service, and every specialized force of employees. All are an essential part of the hospital performance, and members removed from the hospital team by any cause, be it illness or legislative action, weaken our performance, with the weight of lessened service being the patient's reward. All of the work and duties performed at our hospital in contradistinction to

several of the state hospitals are done by salaried employees. Few, if any, of our tuberculous or mentally ill patients are capable of performing in a dependable manner any useful work tasks. Incidentally, we have no volunteer workers as yet at this hospital. In spite of this, we are proud to report that we have recently again received certification by the Joint Commission on Hospital Accreditation, a status which we have enjoyed these past several years but one that cannot be maintained if personnel or operating expense budgets continue to dwindle.

I should like to discuss services available to our patients, beginning with 5 thumbnail sketches of those in the nonmedical category.

Social service. This excellent function is performed by one devoted worker with the assistance of a single clerical employee. This little, but really big, department carries its load with willingness and optimism. Rehabilitation counseling is made available weekly, and such recreational activities as are available to us are coordinated and put on by this "2-cylinder" team.

School. In connection with the State Board of Education, we have a teaching staff of 3 teachers during the normal school year who furnish bedside instruction to patients from the age of 5, sometimes even 4, up to and through high school years. High school certificates of graduation are annually presented. The grade school child and young high school "adult" receive standard courses of instruction and can keep up with their classmates at home.

Church. Fine, faithful, and underpaid, so-called part-time ministers give regular weekly church services in our sanatorium chapel. The bedfast patient may participate through a broadcast system with earphones until he is physically well enough to attend. The ministers all give of their time and make repeated and frequent visits to their sick and needy throughout the week.

Occupational therapy. An under-praised staff comprising one handicraft instructor is responsible for the instruction of all patients capable of participating in this program. The needs are many, the wants diverse, and the instructor has one pair of legs, a similar number of arms, one brain, one pair of eyes, and a low budget with which to work.

Library. We have reason to be proud of our library, which has been built up over the years through the conscientious and diligent services of a registered librarian. Her services were trimmed to meet the needs of all—comics to cosmic science. Unfortunately, this position is vacant, with little hope of filling it, and the position could well be blocked permanently by the

need to "substitute" it by hiring a badly needed nurse in the position.

The following are the medical services which we offer our patients.

Medical staff. All doctors have long experience in the field of tuberculosis and related chest conditions, representing a composite experience in excess of seventy-seven years. This group of physicians comprises the superintendent and staff of three. The superintendent has no directly assigned patient responsibilities. His professional touch on the sanatorium pulse is made largely through regular daily conferences with the medical staff and through personal conferences with the individual physicians about their patients. This superintendent, unlike those in several of the state hospitals, takes his regular duty shift every fourth night and every fourth week end. He is called upon to assist in certain diagnostic procedures and technics and, at times, in performing minor surgical operations. In addition to administrative duties which require the major part of his time, he is involved in the gratis interpretation of large numbers of roentgenograms—more than 10,000 a year—sent in by outside physicians. He is acting director of the Indian Field Service, which requires his presence for purposes of tuberculin testing during the fall and winter months at schools attended by Indian children and includes any and all other responsibilities relating to this service. Annually, he personally directs and performs the tuberculin testing of all schools in Cass County, comprising more than 5,000 students plus adult school personnel and amounting to a total in-county travel in excess of 875 miles. He reviews all correspondence personally, dictates hundreds of communications, and sees most outpatients for chest x-ray interviews. He directs, often alone, entire community tuberculin testing and mobile unit x-ray surveys and assists in others in some capacity at great distances from the sanatorium. Such surveys have been carried out in the communities of Walker, Cass Lake, Remer, Isle, Crosby, and Belgrade. All of the miniature x-rays resulting from these surveys are read by him. He is called upon to attend several official meetings with governmental and departmental officials annually and to address professional groups, lay organizations, and others on the subject of tuberculosis in his *spare* time!

The real producers in the medical staff are those 3 devoted, willing, faithful, and knowledgeable doctors who have assigned to them the various services and are responsible for the entire care and treatment of the patients under their supervision. It should be understood that this

is not custodial care; it involves not only a complete physical examination and history of each admission but also the repeated periodic complete examination and presentation at staff conferences of every patient in the doctor's respective service at intervals of three to six months. The daily rounds, interviews, examinations, special treatments, surgical care, and diagnostic procedures involved in the therapy of the regular hospital inpatient are only a part of the medical load. The medical staff, for instance, examined, advised, diagnosed, and "conferenced" a total of 952 patients during the last year. Part of this load consisted of short-term admissions of 132 new diagnostic cases referred to the sanatorium by outside physicians. The load was further built up by 550 returning or readmitted patients who were completely restudied, involving all aspects of the medical program from laboratory work to dietary planning. And all this is crowded into a three-day, actually an average of sixty hours, program! The medical staff must interview relatives of all patients and is responsible for the complete correspondence pertaining to follow-up and medical summary. All general medical functions such as interpretation of the over 6,000 x-ray films—taken by the hospital, performance of all autopsies, and emergency care of employees, are carried out by the staff. The entire staff, including the superintendent, must cover for each other during vacation periods. All work together as a team.

Laboratory. This staff is comprised of 2 medical technologists, 1 bacteriologist, 1 x-ray technician, and 2 laboratory technicians. The latter are in-service trained. The responsibilities of this group include the scheduled performance of both patient and employee routines, consisting of periodic chest x-ray films, skin tests, blood studies, and urinalyses. Also, during the year, there are hundreds of outpatient chest x-ray films, skin tests, culture examinations, and so forth. The laboratory force must perform blood and urine chemistries and extensive bacteriologic culture preparations and manufacture all laboratory culture media. X-ray service is complete, including plainigraphy, and must also encompass the use of the portable hospital unit as well as the mobile x-ray unit truck. This is a seven-day service. Average annual number of procedures: 7,822 gastric washing cultures, only 7 sent to State Board of Health; 1,688 sputum smears; 2,247 sputum cultures, only 10 sent to State Board of Health; 1,872 urinalyses; 436 urine cultures; 5,688 blood specimens; 1,147 diagnostic skin tests; 110 electrocardiograms; and 6,089 x-ray films.

Nursing department. The nursing staff consists of 4 supervisors, 1 surgical supervisor, and about 70 station nurses, including floor supervisors. Only 11 of this total are registered nurses. Their responsibilities consist of complete and specialized care of both the tuberculous and the senile mentally ill patients on a twenty-four-hour day, seven-day week coverage, complying with Civil Service requirements regarding daily work hours, forty-hour weeks, vacation time, and sick leave. This staff, under these regulations, must be assigned to cover 9 separate patient areas, none of which is physically designed to permit the nurse on duty to scan her patients at a single glance.

Among the problems inherent in this largest of hospital departments and affecting the type and continuity of care available to each patient is the fact that, on an average, there are 1 or 2 nurses on sick leave, often extensive, at all times. Their average age is 45 to 55 years. Two-thirds of the nurses have served in excess of five years and are entitled to fifteen vacation days by Civil Service regulation. Of the total number, 11 have accumulated in excess of one hundred days of sick leave and thus "earn" additional vacation time up to twenty-one days. All of the rest have twelve vacation days annually, and holidays apply to all. These operational details serve to point up the fallacy of attempting to calculate nurse-to-patient ratios by the simple application of "black numbers on white paper."

The foregoing highlights concerning the professional staffs must be set against the kinds of patients to be given care as well as the total annual admissions, which number 1,071, and discharges, which number 1,095. Patients hospitalized in the Minnesota State Sanatorium are of both sexes and range in age from infancy to senility. Incidentally, during the past twelve months, we have admitted 36 children and have readmitted for follow-up evaluation 106 others.

The ingredients of our hospital's patient "recipe" include the white and the Indian, the patient of normal mentality, the patient with mental illness and deterioration, the tuberculous, and the nontuberculous. The need to keep separate these various categories is evident. Many of our patients are bedridden and require total body-function care. All of this care by this staff is done, to repeat, without patient workers or organized volunteers. All employee services are governed strictly by Civil Service regulations. All of this service is performed and furnished at a current per diem rate, calculated by our own Department of Public Welfare, of \$11.14. All such receipts, plus others for service, are

turned in directly to general revenue.

Dietary department. This staff is second largest in the hospital and consists of 1 registered dietitian, 1 assistant dietitian, cooks, special diet supervisors, a meat cutter, a baker, ward-kitchen maids, and serving personnel in both patients' and employees' dining rooms. Their responsibilities include the preparation and serving of approximately 1,000 meals daily and the management of two separate dining room areas—one for ambulatory patients and the other for employees. Special diets must cater to the needs of infant children and edentulous senile patients unable to feed themselves, to say nothing of the daily medical diets needed in the treatment of the diabetic, the ulcer, and the "touchy gall-bladder" patients.

Pharmacy. Part-time service is provided six days a week by a registered pharmacist recruited from the nearby town.

Special professional consultants. These persons are "employed" under special contract on a visiting basis and give consultation and perform surgical procedures in the fields of dentistry and ophthalmology and general, thoracic, and orthopedic surgery.

Indian Field Service. This program is centered in the sanatorium and provides, under contract between the Department of Public Welfare and the United States Public Health Service, for a medical director, a field nurse, a social worker, and a clerk-stenographer. The area under supervision principally includes the Leech Lake, Cass Lake, Red Lake, White Earth, and Mille Lacs reservations. Other, even more remote, areas where Indian people reside are also given tuberculosis case-finding, hospital treatment, and follow-up care. One member of the Minnesota State Sanatorium medical staff is responsible for the interpretation of all chest x-ray films taken of Indian patients in the various Indian hospitals and clinics—2,914 films during the past year. Nearly 5,000 tuberculin skin tests were applied in schools, clinics, and homes in the last year.

Sanatorium outpatient services. No special or separate department functions in this respect. Chest x-ray films and tuberculin skin tests are available to all persons referred by physicians and to all inpatient contacts and family members. Interpretations are made and submitted to the referring physicians, to public health nurses, and to the State Board of Health. More than 10,000 chest x-ray films taken and submitted by doctors, clinics, and hospitals from all parts of the state are interpreted annually. Routine chest x-ray films of all positive tuberculin reactors found in school surveys in several surrounding counties are taken and interpreted.

RECOMMENDATIONS

1. Intensify rather than slacken tuberculosis case-finding at all levels in Minnesota.

2. Carry out fuller instruction in the diagnosis of tuberculosis in our medical schools in formal curriculum and in postgraduate courses.

3. Inculcate respect for the infectiousness of this disease and its results from the beginning of training in medical education and carry it on into the field of medical practice.

4. Place emphasis upon the legal obligation of the medical profession to report tuberculosis as an infectious disease to the public health authorities in our state, and, as a corollary, place emphasis upon the moral obligation of the medical profession to report and insure isolation of the active tuberculous patient as a safeguard to our citizenry, not in our homes, stores, streets, schools, and business places but in sanatoriums created to serve this function.

5. In return for fifty-two years of dedicated service in battling this deadly, insidious, and unpredictable disease, the Minnesota State Sanatorium should be given the support, the backing, and the inspiration it has earned and deserves from its governing body and related agencies so that it can continue its efforts at control and eradication; it should not be let down and cast aside, creating in the staff a sense of blank defeat and frustration.

CIGARETTE SMOKING appears to be an important factor in the development of idiopathic obstructive emphysema. Maximal expiratory flow rates (MEF), used as a screening test for emphysema, show little change before the age of 70 in nonsmokers. However, the decline begins in the mid 30's among smokers and is generally greater in heavy than in light smokers.

A. L. FLICK and R. R. PATON: Obstructive emphysema in cigarette smokers. Arch. Int. Med. 104: 518-526, 1960.

A Tuberculin Survey of Migrant Workers, 1959 A Prototype for a State-wide Program

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FOR MANY YEARS, migrant workers coming to Minnesota have been considered a source of infectious disease, threatening the health of the local population. However, little factual data to support this feeling has ever accumulated in the files of the State Health Department. Beyond isolated reports of ill health in the migrant families themselves, there has been little evidence that infections in the local community could be traced to the migrants.

Many difficulties have hindered the normal procedures that would help to clear up this confusion as well as to hasten the assimilation of these migrants into normal community patterns. Language, legal residence requirements, and, especially, the migratory nature of their work and existence have all constituted barriers tending to set migrants apart from usual community case finding and case treatment routines.

One way to overcome these difficulties is to try out a well-planned technic in a local setting, which, if successful, can then be utilized in other localities. Eventually a state-wide program can be erected on this foundation of procedures, each tailored to fit the local situation. In the field of tuberculosis control, the tuberculin survey of migrants at Hollandale in 1959 provides the model on which to begin building an effective state-wide program.

PROCEDURE

In Minnesota, effective tuberculosis control programs must be based primarily upon cooperation and utilization of local facilities and resources, with the help of additional services from both official and voluntary state agencies. A successful program must be acceptable to the persons to whom it is directed as reasonable and worth the cost in time and money required.

Surveys, such as that at Hollandale, involve 3 stages: planning, the survey itself, and the follow-up. The third stage is the most impor-

tant of the three, and unless follow-up can be adequately carried out, it is wasteful and frustrating to engage in a survey. Doing something about the positive findings constitutes the follow-up stage, and usually this is the hard work of the survey. Unless it can be accomplished, however, little has been gained.

Early in June 1959, the Minnesota State Employment Service requested that a tuberculosis survey be carried out among migrant workers in the Hollandale area of Freeborn County in southern Minnesota. After a rapid series of organization meetings and field visits, a tuberculin testing survey with 70 mm. photofluorograms of all positive reactors was carried out from June 22 through June 25. Chest x-ray films, 14 by 17 in., were taken of those individuals whose survey films showed suspicious findings. In presurvey registration, 907 persons were given appointments for tests; 558 participated, including 469 migrant workers and 89 local residents (tables 1 and 2). Two hundred thirty-one persons had 70 mm. photofluorograms, and 43 required 14 by 17 in. x-ray films. The organizations participating included, besides the state and local employment services, the Minnesota and Freeborn County tuberculosis and health associations, the Minnesota Department of Health, the Minnesota Department of Welfare, the Mineral Springs Sanatorium, the Freeborn County public health nurse, the Freeborn County Welfare Board, physicians of the Freeborn County Medical Society, ladies-aid societies of four local churches, and others.

RESULTS

The Hollandale survey provides valuable data to help decide the seriousness of the tuberculosis problem in migrants as a special group of the population. As tuberculosis declines as a health problem in the general population, control efforts must be concentrated on those groups in which the disease continues to be significant.

In 1919, 373 children in Minnesota under 20 years of age died of tuberculosis. In 1949, a

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TABLE 1
MINNESOTA DEPARTMENT OF HEALTH
REPORT OF GROUP TUBERCULIN TESTS

County: Freeborn		Date of Test: June 22-23, 1959			
School or Center: Hollandale (Mexican Migrants)		Strength: 1:1000			
Type of Tuberculin used: O.T.		Strength: 1:1000			
Age (Yrs.)	Male		Female		Total
	Negative	Positive	Negative	Positive	
(1)	(2)	(3)	(4)	(5)	(6)
Under 1	3		5		8
1	1		8		9
2	8		6		14
3	4	2	10		16
4	9	1	5		15
5	8		5	1	14
6	12		3		15
7	4	1	7		12
8	9		5	2	16
9	9		9	1	19
10	4		8	1	13
11	10	4	8	1	23
12	5	1	8		14
13	9	3	9	1	22
14	6	2	9	2	19
15-19	22	18	23	8	71
20-24	5	15	6	4	30
25-29	1	9	5	4	19
30-39	5	18	3	18	44
40-49	1	17	2	14	34
50-59	4	12	3	6	25
60 and above		8		7	15
Total	139	111	147	70	467

total of 15 children under 20 years of age died of this disease. In 1958, only 1 death, in an Indian girl 18 years old, occurred in persons under 30 years of age. The total state rate for tuberculosis deaths for all ages has consistently been reduced over the past forty-year period. In the ten-year period from 1949 through 1958, for example, the rate dropped from 13.6 per 100,000 population to 3.1.

Among children, tuberculosis has been practically eradicated. In Minneapolis in 1926, 47.3 per cent of children from kindergarten through eighth grade reacted positively to tuberculin tests. By 1936, the percentage had been reduced to 18 per cent; in 1944, only 7.7 per cent reacted; and, in 1954, only 3.9 per cent of 11,976 children tested in the same 24 schools had positive tuberculin tests. In figures gathered on a state-wide basis in 1958, there were 3.12 per cent positive reactors out of 192,434 persons of all ages tested. Only 0.90 per cent of 21,965 6-year-old children reacted positively, and only 2.89 per cent of 16,532 14-year-old children were positive.

Results of the Hollandale survey indicate, however, that tuberculosis remains a serious problem in this migrant group of the population. In the 467 Texas-Mexican migrants surveyed,

TABLE 2
MINNESOTA DEPARTMENT OF HEALTH
REPORT OF GROUP TUBERCULIN TESTS

County: Freeborn		Date of Test: June 22-23, 1959			
School or Center: Hollandale (Permanent Residents)		Strength: 1:1000			
Type of Tuberculin used: O.T.		Strength: 1:1000			
Age (Yrs.)	Male		Female		Total
	Negative	Positive	Negative	Positive	
(1)	(2)	(3)	(4)	(5)	(6)
Under 1			1		1
1					
2	1				1
3			2		2
4	2		2		4
5	2				2
6	1		1		2
7					
8			1		1
9	2		3		5
10	2		2		4
11	1		2		3
12				1	1
13			1		1
14	1		1		2
15-19					
20-24		1	1		2
25-29	2	1	4		7
30-39	10	2	10	3	25
40-49	3	3	5	1	12
50-59	2	2	2	1	7
60 and above	1	3			4
Total	30	12	38	6	86

238 persons were 15 years of age or older, and 158, or 66.4 per cent, of this group reacted positively to the tuberculin test. This was compared with 1958 data for the general population of Minnesota, which showed that only 13 per cent of adults 30 years old and 36 per cent of adults 65 years or older were positive reactors.

In the migrant workers' survey, 229 children under age 15 were tested, and 23, or 10 per cent, were positive reactors.

The migrants have far more tuberculosis, as shown by Mantoux tests, than do the Indians in Minnesota.

Photofluorograms, 70 mm., of the 181 positive reactors were taken at the time the tuberculin test was read. Forty-three of these persons were then advised to have regular 14 by 17 in. chest x-ray films because of suspicious findings in the survey films. Thus, over 9 per cent of the entire group of 467 migrants, or almost 24 per cent of the 181 positive skin reactors, required large chest roentgenograms.

Experience with the Hollandale survey revealed certain problems that require special emphasis. First, planning for a survey must begin early enough to allow time for necessary arrangements. Standardized and simplified procedures and record forms should be utilized.

There must be adequate, trusted individuals available to serve as interpreters and to insure good communication with the migrant families. Thorough clearance of the program with the local medical group and with the county welfare board director is important, especially for follow-up arrangements. A local public health nurse who can direct the follow-up and centralize the coordination and responsibility for the entire survey is the best guarantee of success.

The accomplishments in attacking tuberculosis

in this group of the population in the Hollandale area illustrate what can be done through effective organization of public and private health services directed against a particular disease. This program can be duplicated and adapted to fit the local situation in any similar locality where migrant workers gather in Minnesota. It could be repeated annually to screen this population group as soon as they arrive in the state. Because the program uses existing facilities, it is a practical approach to a difficult problem.

ATYPICAL ACID-FAST bacilli differ from *Mycobacterium tuberculosis* in culture characteristics and in failure to produce progressive disease in guinea pigs. Since the majority of patients with atypical infections have not been given antituberculous drugs, these organisms are not thought to be tubercle bacilli altered by drug therapy. Three different types of atypical mycobacteria have been identified by pigment production upon exposure to light. The photochromogens produce yellow pigment when grown in the presence of light and white or buff colonies when grown in the dark. Most scotochromogens are yellow-orange, whether grown in the light or the dark, and the nonchromogens or Battey type produce white, buff, or yellowish pigment under both light and dark conditions. All 3 types grow on culture media in six to forty-two days at room temperature. *Mycobacterium tuberculosis* will not grow at room temperature, but *Mycobacterium fortium* and nonpathogenic saprophytic acid-fast bacilli will grow within three days at room temperature.

The photochromogens, a closely related group, more closely resemble the tubercle bacilli than do the other atypical organisms. Disease produced by the photochromogens is most prevalent in the northern central states. The scotochromogens are nonpathogenic for laboratory animals and are more closely related to the saprophytes. The majority of nonchromogen cases have been reported in the southeastern portion of the United States; the nonchromogens are intermediate in pathogenicity between the photochromogens and the scotochromogens.

Most patients with atypical infections react positively to intermediate PPD intradermally. Epidemiologic studies show a lower incidence of abnormal chest film and tuberculin skin tests in contacts than would be expected. However, atypical PPD has not been used in skin testing.

Of patients admitted to Florida state tuberculosis hospitals, 2 per cent have atypical mycobacteria infection. Of 100 patients with atypical infections, 7 were photochromogen; 15, scotochromogen; and 78, nonchromogen. Infection was seen predominantly in elderly white males from rural areas. Pulmonary disease appeared to be caused by atypical mycobacteria in 74 of the patients; the other 26 had either proved tuberculosis or single isolations of atypical mycobacteria. Of a total of 10 deaths, only one was due solely to progressive atypical infection. In spite of treatment, 48 patients still show mycobacteria in sputum.

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Clinical Application of Pacatal in Tuberculous Patients

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EMOTIONAL DISTURBANCES often precede and contribute significantly to the development of tuberculosis, an observation that is even more evident in cases of relapse.¹ Indeed, many investigators postulate that illness may develop as an escape from frustration and the responsibilities of life or as a method of self-punishment. Tuberculosis patients frequently suffer from many psychiatric conditions and psychiatric symptoms. Chief among these are anxiety, tension, agitation, restlessness, and insomnia. Therapy, therefore, should be directed toward reducing their conflicts and controlling overactivity and anxiety by psychotherapy, physiotherapy, and appropriate relaxing drugs. The use of neuroleptic drugs plays a part in this. Among others, Pacatal was tested on selected patients in this hospital for a period of four to eight months.

PHARMACOLOGY AND CLINICAL ACTION

Pacatal (N-methyl-piperidyl-(3)-methyl phenothiazine), a substituted phenothiazine compound, was synthesized by Schuler and Nezel. The detailed pharmacology of this drug has been described elsewhere.² Early clinical investigations were published in Europe on the drug's analgesic and anesthetic preoperative effects and its action in deep sleep. It was used as premedication in general anesthesia and as a drug for potentiation of hibernation anesthesia. A fall of the basal metabolic rate and blood sugar level without a simultaneous fall of blood pressure, pulse, or temperature was found in these studies.

In the human being, it has both parasympatholytic and sympatholytic actions with a predominance of the former. It, therefore, has a marked atropine-like effect. This latter includes inhibition of secretion and mydriasis. In addition, it has a pronounced energy reducing action with-

out, however, a marked effect on blood pressure, so that patients tend to be able to remain ambulatory.

Its psychiatric use and the major neuroleptic effects are well described in the paper by Sarwer-Foner and Koranyi.³ Its main actions in this regard, as documented by them, are as follows: It tends to reduce energy output, that is to say, the energy available for muscular energy when the drug is given in adequate dosage. It slows down and sedates many patients—many feel tired, weak, and, occasionally, dizzy. It stops motor overactivity. It leaves the sensorium clear at all doses. It does not seem to change effecting impulses per se but tends to lessen the energy available for translating the impulses into action, as compared to thought, if the dose is adequate. Appetite seems good in most patients on this drug.

It can improve the patient's ability to sleep at night, especially after the first few days, although it does not have a marked hypnotic effect. It makes some patients moderately drowsy, especially if it is given intramuscularly. Patients can fight sleep when it is induced. It does not seem to have any significant changes in pulse, blood pressure and respiration in the doses used by Sarwer-Foner and Koranyi.

USE IN TUBERCULOUS PATIENTS

Because of the influence of the patient's total personality on his illness and the way in which he responds to hospitalization and bed rest, it is now a routine procedure at the Mount Sinai Sanatorium to assess every new patient from a psychiatric point of view. Many cases have been found that require active treatment to control psychiatric illnesses. This is an integral part of treating their tuberculosis.

Apart from psychiatric illness or symptoms, many tuberculous patients often have night sweats and insomnia as well, and, because of the atropine-like action and drying effect of this drug, it was decided to treat some of these patients with Pacatal as well.

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TABLE 1
PSYCHIATRIC DIAGNOSES IN GROUP A1

Name	Sex	Age	Psychiatric diagnosis
H.D.	F	29	Anxiety reaction in a paranoid personality.
M.S.	F	41	Anxiety reaction with marked somatization
J.R.	M	33	Anxiety in an obsessive-compulsive personality
Z.K.	M	39	Depressive reaction with anxiety features
A.M.	M	55	Depressive reaction
E.S.	M	43	Phobic reaction
H.L.	F	41	Anxiety reaction with marked somatization
L.M.	M	22	Schizophrenic reaction, paranoid type
A.C.	M	56	Schizophrenic reaction, paranoid type
J.K.	M	33	Anxiety reaction in a schizoid personality
F.S.	M	47	Anxiety reaction in a schizoid personality
N.B.	M	49	Depressive reaction
M.N.	F	45	Anxiety reaction in an obsessive-compulsive personality
C.N.	F	33	Anxiety with somatization
D.R.	M	47	Anxiety reaction in a paranoid personality

In certain personality patterns, the prognosis of tuberculosis becomes grave because of the patient's inability to tolerate the hospital regimen. Behavior in the hospital is often consistent with earlier behavior patterns, and knowledge of the patient's personal history is, therefore, useful for prognosis and practical management.⁴

Three groups of patients were given Pacatal. Group A1 consisted of tuberculous patients with psychiatric illness as well. Group A2 was comprised of tuberculous patients with night sweats and insomnia but without major psychiatric illnesses or disturbances. Group B was made up of a small number of employees and members of the staff who were nontuberculous but who were suffering from psychiatric illnesses necessitating the use of tranquilizers. This latter group had

symptoms, such as anxiety, agitation, motor restlessness, insomnia, night sweats, tremor, and, in some cases, depression.

Night sweats can be beneficially influenced by atropine because of this drug's action on secretory glands innervated by postganglionic cholinergic nerves. Unfortunately, the action of atropine is not selective, and side effects are frequently encountered affecting the central nervous system, the respiratory tract, the heart and circulation, and also the gastrointestinal system. The symptoms in patients taking atropine are dryness of the mouth, blurred vision, weak and rapid pulse, urinary urgency, restlessness, confusion, and mania or delirium. Since previous clinical investigations have demonstrated many atropine-like actions of Pacatal, it was decided to try the drug on patients complaining of excessive perspiration and night sweats.

Twenty-six cases were studied. The group consisted of 18 men and 8 women ranging in age from 20 to 75 years, with an average age of 42 years. The 26 patients were divided into three groups: A1, the first group consisted of patients with tuberculosis who received treatment with different antibiotics. Pacatal was given to reduce psychomotor drive and muscular energy, to help sleep, and to reduce anxiety which occurred secondarily to the control of the aforementioned symptoms. The second group, B, consisted of employees who were prescribed Pacatal because they required a neuroleptic medication. The third group, A2, also tuberculous patients, took the medication in an attempt to control night sweats and insomnia, but they did not have major psychiatric symptoms.

TABLE 2
TUBERCULOSIS DIAGNOSES IN GROUP A1

Name	Tuberculosis	Sputum	Duration of disease
H.D.	Genitourinary tuberculosis	Negative	1 year
M.S.	Minimal pulmonary tuberculosis	Positive	1 year
J.R.	Moderately advanced pulmonary tuberculosis	Positive	6 months
Z.K.	Moderately advanced pulmonary tuberculosis	Negative	1 year
A.M.	Moderately advanced pulmonary tuberculosis	Positive	1 year
E.S.	Minimal pulmonary tuberculosis	Negative	2 years
H.L.	Moderately advanced pulmonary tuberculosis	Negative	5 years
L.M.	Plenisy	Negative	6 months
A.C.	Far advanced pulmonary tuberculosis	Negative	10 years
J.K.	Moderately advanced pulmonary tuberculosis	Negative	4 years
F.S.	Moderately advanced pulmonary tuberculosis	Negative	6 months
N.B.	Moderately advanced pulmonary tuberculosis	Positive	6 months
M.N.	Minimal pulmonary tuberculosis	Negative	2 years
C.N.	Far advanced pulmonary tuberculosis	Positive	6 years
D.R.	Far advanced pulmonary tuberculosis	Negative	20 years

The dose of Pacatal was 25 mg. three times daily and at night. The study was conducted for four months or longer, with a maximum of eight months. Appropriate screening tests were done on blood, urine, and liver to determine any drug toxicity. The patients were psychiatrically assessed by the psychiatrist member of the team before being treated with the drug. The patient's progress was followed daily by the medical, nursing, and occupational therapy staff, and the psychiatrist saw the patient and followed his progress weekly. Assessment of the progress made in treating the tuberculosis was done by the roentgenogram, bacteriologic methods, and physical examinations. Particular attention was given to any demonstrable effect of Pacatal and the concomitant antituberculosis drug therapy.

RESULTS

Group A1 (tables 1 and 2) of 10 men and 5 women consisted of patients with tuberculosis and psychiatric illness. Table 1 shows the detailed psychiatric diagnoses and table 2 the tuberculosis diagnoses. All of these patients had proved tuberculosis. All had received anti-tuberculosis antibiotics prior to Pacatal medication. All had severe emotional disturbances with symptoms which made bed rest difficult and hampered adjustment to the hospital routine, such as anxiety, increased restlessness, increased motor drive, phobic and paranoid reactions, increased aggression, insomnia, and poor appetite with depressive features in some cases. It had, therefore, been felt that the psychologic factors were hindering many of these patients' progress. Of the patients listed in tables 1 and 2, 13 of the 15 were discharged six to eight months after Pacatal had been started. All of them had been treated for their tuberculous conditions for longer than four months prior to Pacatal therapy. The average stay of the patient in Mount Sinai Sanatorium is eleven months, and, despite the initial lack of therapeutic response in the first four months prior to the administration of Pacatal, these 13 patients approximated this average figure at the time of their discharge after being placed on Pacatal.

Favorable progress on the chest x-ray could be demonstrated in all of these cases, and this improvement appeared to be more striking than it was before Pacatal was begun. Rigid bed rest periods were more easily observed after Pacatal was initiated and with less resistance. These patients also more readily accepted the regular anti-tuberculosis drug administration. Many experts in chest diseases still believe in prolonged rest, and it is almost the consensus of opinion that at

TABLE 3

Name Group A1	Weight gain during 4 months of Pacatal therapy	—Monthly average—	
		Before Pacatal	During Pacatal
H.D.	13	2	3½
M.S.	16	2	4
J.R.	13	2½	3½
Z.K.	12	0	3
A.M.	20	2½	5
E.S.	16	1	4
H.L.	7	0	1½
L.M.	8	0	2
A.C.	1	0	¾
J.K.	6	0	1½
F.S.	20	2	5
N.B.	6	1	1½
M.N.	10	1	2½
C.N.	4	0	1
D.R.	0	0	0

least the first three to six months should be spent in a sanatorium with adequate rest facilities.

Successful reduction of energy output was observed in all patients, as previously described by Sarwer-Foner and Koranyi, when the dose level was adequate for this purpose. After treatment with Pacatal was started, improvement in the patient's psychiatric complaints soon became evident. Psychomotor excitation and overactivity was relieved, with the energy channeled into more controllable activities, such as occupational therapy. Concomitantly with this, anxiety usually lessened. Eleven of the 15 patients commenced diversional activities shortly after starting to take Pacatal. Insomnia lessened in all, and, in 6 cases, we were able to discontinue hypnotics. Appetite had generally increased, and the average weight gain was over 10 lb., occurring somewhat more rapidly than the average weight gain prior to Pacatal administration (table 3).

Group B is shown in table 4 and consisted of employees who were placed on Pacatal to alle-

TABLE 4
DIAGNOSES IN EMPLOYEE GROUP B

Name	Sex	Age	Psychiatric diagnosis
S.P.	M	26	Anxiety reaction in an obsessive-compulsive personality
G.O.	F	32	Anxiety reaction with marked somatization
G.K.	M	42	Anxiety reaction with marked somatization
J.B.	M	50	Schizophrenic reaction, paranoid type

TABLE 5
PSYCHIATRIC DIAGNOSES AND SYMPTOMATOLOGY
GROUPS A1 AND B

Depressive reactions	3
Phobic reactions	1
Anxiety reactions:	
In paranoid personality	2
In obsessive-compulsive personality	3
In a schizoid personality	2
With somatization	5
Schizophrenic reactions, paranoid type	3
	19

viate psychiatric symptoms. Of this group, 3 were men and 1 was a woman.

Our psychiatric results were thus very similar to those found in open psychiatric settings.¹ Psychomotor excitation and overactivity were controlled, and the patients became calmer. Energy output lessened, sleep improved, appetite increased, and anxiety was greatly reduced in many patients. They felt slowed, weak, and occasionally dizzy. Good effects on complexes, such as depression and anxiety, were produced by a combination of factors and not by the action of the drug alone. This latter was no different than with other neuroleptic agents. A summary of the psychiatric diagnoses and symptomatology of groups A1 and B is presented in table 5.

Agitation was common and insomnia almost the rule. One of these patients suffered from anxiety with depression and became more depressed on the drug and subsequently had to be given electroshock therapy because of suicidal tendencies. Two other patients were schizophrenics of the paranoid type and had to be committed to closed psychiatric wards. One of them was kept at the sanatorium on heavy doses of Pacatal for about two weeks, during which time he became fairly well controlled and manageable. Although

his agitation was controlled, his general mental state still made treatment in a closed psychiatric hospital desirable.

All patients in groups A1 and B felt that they were helped by the drug except for the 2 patients just mentioned. The striking differences between the tuberculous and the nontuberculous groups were seen in the side effects. None of the tuberculous patients complained of any undesirable sensations. It was evident that tuberculous patients tolerated the drug much better than the employees who did not have tuberculosis. All of the staff members showed varying degrees of dryness of mouth, irritable throat, and occasional blurred vision. This again was similar to what had been described in earlier papers^{2, 3} in psychiatry. None of these complaints were severe enough to force us to discontinue Pacatal in this series, but the differences between the patients and staff were quite prominent in this regard.

This experience suggested that the side effects in tuberculous patients are much less because the patients benefited from the atropine-like action of the drug and liked it. We, therefore, selected 7 patients who complained of excessive perspiration and night sweats to whom to administer the drug. As mentioned before, these patients did not have any psychiatric symptoms. Table 6 shows the group A2 patients, the tuberculous patients without psychiatric conditions given Pacatal for night sweats and insomnia.

Most of these cases were chronic patients with advanced lung disease and toxic symptoms. The results in this group were more encouraging. The night sweats, which made the patients very uncomfortable and even interfered with their sleep in 4 of the cases, completely disappeared. In others, they decreased to a great extent. The nurses stated that 4 of the 7 patients in group A2 previously had night perspiration requiring special attention, and most of them ceased complaining shortly after medication was started. All 7 patients in this group had been taking nighttime

TABLE 6
GROUP A2

Name	Sex	Age	Diagnosis	Duration of disease	Sputum
A.R.	F	23	Pulmonary tuberculosis, minimal	2 years	Positive
C.D.	F	34	Pulmonary tuberculosis, far advanced	20 years	Positive
J.H.	M	54	Pulmonary tuberculosis, far advanced	4 years	Positive
R.H.	M	27	Pulmonary tuberculosis, far advanced	7 years	Positive
W.P.	M	75	Pulmonary tuberculosis, minimal	5 years	Positive
H.H.	M	25	Pulmonary tuberculosis, far advanced	2 years	Positive
L.K.	M	53	Pulmonary tuberculosis, moderately advanced	15 years	Positive

sedation for insomnia, which was quickly discontinued after the institution of Pacatal. Only 1 patient complained of dryness of the mouth and felt some discomfort. In this case, the drug was discontinued shortly after the patient started to complain. The remainder showed favorable responses, and their general well-being mounted as their morale improved.

The behavior of the tuberculous lesions was not expected to change much because of the chronic nature of the disease. Surprisingly, roentgenograms showed improvement in 2 of the very ill patients, and 1 of these had conversion of the sputum. One of these patients, who was 75 years of age, tolerated the drug very well and did not present any of the side effects mentioned in previous reports with elderly patients.

SIDE EFFECTS AND TOXICITY

Side effects were seldom noticed, and, except for the single patient we previously referred to with dryness of mouth, there were no complaints from the two groups of patients. In the group of staff members, one stated that she felt very dizzy during the first few days she took the drug but that subsequently the dizziness disappeared, and she continued to take Pacatal without any untoward effects. This staff member had a great deal of somatic complaints, and it is possible that her complaints were due to other than drug factors. In general, the group of employees complained more of dyspepsia, dry cough, and dry throat than the 2 groups of tuberculous patients.

There were no other side effects nor did we encounter any toxic effects, even after eight

months of administration in some cases. None of the toxic effects reported in some of the previous papers were found in these groups. Mention should be made that some of the patients suffered from diseases such as syphilis, kidney tuberculosis, and cardiac failure as well. Two patients had proved peptic ulcer, and, in these cases, the drug produced an improvement not only in the patient's general well-being but also decreased the complaints regarding hyperacidity and stomach pain.

In chronic cases of tuberculosis, any new drug frequently gives new hope to the patient and may produce a temporary state of euphoria.⁵ This occurred in a few patients but rapidly disappeared as the drug continued to be taken.

There were no significant changes in pulse, blood pressure, or respiration in this series. Although nighttime sleep increased, there was no marked drug-induced drowsiness or hypnosis in the daytime.

CASE REPORTS

J.R., a 33-year-old Rumanian born salesman, was hospitalized for pulmonary tuberculosis in August 1956. Other than his feeling that adolescent masturbation was incorrect and harmful, there was nothing particularly contributory in his early history. At age 18, he was in a Nazi concentration camp where he suffered the usual abuse. In addition to this, he was once struck by an SS soldier with a rifle in the right frontal region and lost consciousness. He had complained of headaches since then. After the war, having survived the concentration camp, he married and apparently had no particular marital difficulty. His pulmonary disease was detected at the end of July 1956 when his sputum became positive, and he was admitted to the Sanatorium. Antibiotics were started, but the patient's condition seemed to progress

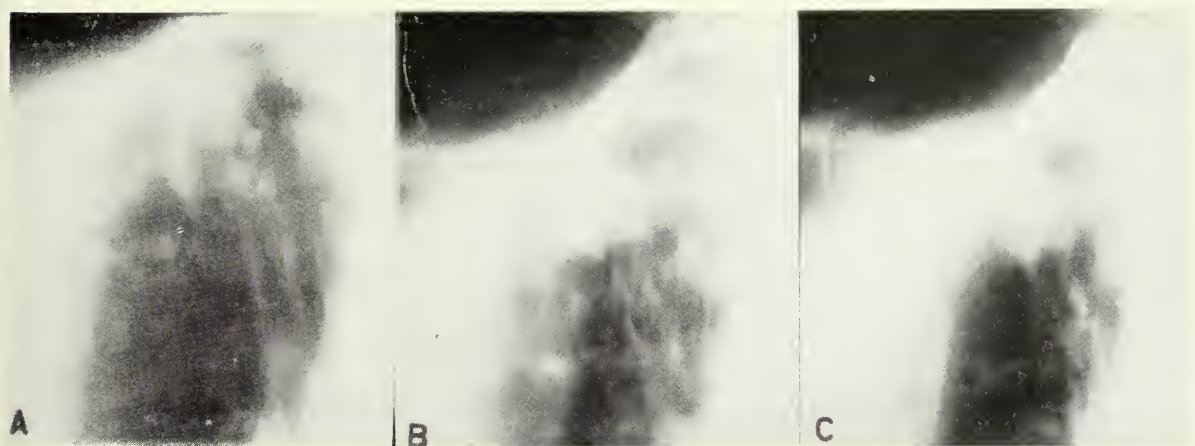


Fig. 1. Tomograms of the right upper lobe of J.R. (group A1). (a) Tomogram taken July 1956, when treatment with antibiotics was started. (b) Tomogram taken February 1957, when Pacatal was started shows still evidence of cavitation. (c) Tomogram taken three months later shows no evidence of cavitation.



Fig. 2. Chest x-rays of C.D. (group A2) taken in August 1957. (a) Far advanced bilateral disease. Patient was placed on Pacatal. (b) Chest x-ray taken eight months later shows increase in the shadows and in the fluid in the left chest. In spite of progress of the disease, the patient's general well-being improved.

very slowly because of extreme restlessness, inability to tolerate bed rest, anxiety, and increased motor activity.

Physical examination showed a well-nourished, normally developed white man who was in no distress. The right upper chest showed dullness with pathologic sounds; otherwise, physical examination was negative. An electroencephalogram was negative as were other laboratory data. When seen by the psychiatrist because of the restlessness, motor symptoms, and anxiety, it was suggested that he should try diversional activities or studies. He refused this suggestion.

The patient was placed on Pacatal in February 1957, and, shortly afterward, his appetite increased tremendously and his general well-being became decidedly improved. At this point, he said that he wanted to take a correspondence course in drafting. He took this course and did very well.

Chest x-rays (figure 1), which until then had shown only very slow clearing, at this point began to show much faster resolution, and his sputum became negative for tuberculosis on smear and culture. His complaints had greatly diminished, and he was discharged on June 1, 1957, in a very good clinical condition.

This obsessive-compulsive patient with marked anxiety and threats to his masculinity by the induced passivity and bed rest, with moderately advanced bacillary pulmonary tuberculosis, had greatly benefited from Pacatal. The control of his restlessness and psychiatric symptoms permitted better participation in the anti-tuberculosis therapeutic regimen. Follow-up shows that

presently, eighteen months post discharge, he seems to be well adjusted, holds a good position, and is supporting his family. The disease in his chest is inactive, and there is no evidence of any active infiltration on chest x-ray.

C.D., a 35-year-old woman, is quoted as an example of the help Pacatal can give to patients without evidence of a psychiatric condition but with night sweats and insomnia.

This patient became ill at the age of 22 in 1943 and was then admitted to a hospital. She was treated with a right pneumothorax and returned home in 1944 to care for her sister who was ill with tuberculosis. The patient's tuberculosis recurred again in 1948, and she was hospitalized in different sanatoriums until June 1955. She was treated with left pneumothorax, pneumoperitoneum, and antibiotics. In 1955, she left the hospital and stayed at home until October 1956, when she was admitted to Mount Sinai Sanatorium. An admission chest x-ray confirmed far advanced, bilateral disease with numerous large cavities bilaterally (figure 2).

Physical examination showed an undernourished white woman with frequent cough, wheezing, and complaints of chest pain and night sweats. There was toxicity with fever, general malaise, and weakness. During the first year of her stay at Mount Sinai Sanatorium, this patient received all available antibiotics without any appreciable improvement in her condition. When seen by our consultant psychiatrist, there was no evidence of any abnormal mental patterns, and, considering her longstand-

ing disease, this patient's spirits and morale were quite good. The only problem seemed to be in reducing the symptoms of her toxicity and the irritative phenomena associated with secretion and cough. Unfortunately, the antibiotics did not produce the expected improvement, and the patient's condition continued to deteriorate.

Pacatal was instituted, and, shortly afterward, the patient's well-being improved. The disturbing night sweats disappeared, as did the constantly clammy skin. The patient felt so much better that, under the guidance of the occupational therapist, she began handicrafts which not only helped her occupy her mind but also helped her financially. She is still on Pacatal after eight months. She seems much less toxic, she has gained weight, and her cough has decreased considerably. Her night sweats have also been improved considerably.

The other members of this group of patients have also experienced similar improvement and show more faith and hope toward the future as a result. These patients with chronic cases who must stay in the sanatorium for many years and who are possibly expecting some new miraculous cure have to be given all the supportive therapy that can be offered to make their lives more enjoyable.

DISCUSSION AND CONCLUSIONS

The comprehensive management of tuberculosis must consider the total person and not only his physical diseases.⁶

The relationship between emotional factors and tuberculosis is a serious problem and constitutes a vicious circle in many cases seen at the sanatorium. Breuer,⁴ who investigated the psychic element in the etiology of tuberculosis, found that there exists a close relationship. Accordingly, when the emotionally labile individual contracts tuberculosis, the emotional factors increase in severity and contribute to a further decrease in his resistance against tuberculosis and an increase in anxiety against accepting treatment.

In many cases of tuberculosis, the cause of physiologic disturbance is the abnormal situation in which the patient finds himself, his regime of life, and the threats to his life and future.⁵ It has been the practice in these cases to break the vicious circle by attacking only the tuberculosis with the aid of antibiotics and chemotherapy. Such cases may improve more rapidly if both problems — tuberculosis and emotional — are attacked at the same time in every case in which this is feasible.

While not every tuberculous patient has mental problems, recent progress in stress research established the fact that stress, which may not be psychic in nature, can be conducive to lower resistance.⁷ Selye showed that this factor plays an increased role in tuberculosis.

We have also noted that alcoholic bouts which often create serious disturbances in many sanato-

ria are due to increased energy and the patient's feeling of hostility toward his surroundings and his disease. Pacatal seems to help some of these patients by controlling selected symptoms without causing the addiction that is seen with some other drugs.

Other types of tranquilizers have been tried in tuberculous patients with fair results. It always has been a problem to select the proper drug because of the general toxicity shown by many of these patients. In this series, Pacatal was found to be without toxic effects, even in patients with far advanced pulmonary disease, in the doses used.

Since isoniazid has been introduced in the treatment of tuberculosis, patients taking this drug have increasingly complained of restlessness, nightmares, and peripheral neural excitability.⁸ Many patients on the high dosage of isoniazid presently employed show signs of increased energy. In them, Pacatal seems to have a beneficial effect.

Patients with tuberculosis often suffer from chronic pulmonary emphysema. In these cases, a sedative without a depressive effect on the respiration is desirable. In our experience, Pacatal did not produce any untoward effect on respiration and can be used safely in these cases.

SUMMARY

This study suggests that Pacatal has a beneficial effect on tuberculous patients showing specific psychiatric symptoms requiring neuroleptic medication. It is particularly suitable for such cases because, despite its excellent neuroleptic effect, its hypotensive and other actions are not marked enough to seriously inconvenience the patients in terms of motoricity when this is not desirable. Patients can remain ambulatory when this is desired.

Side effects of dryness of mouth and other atropine-like actions with decreased secretion are particularly suitable for tuberculous patients in whom night sweats, cough, and the production of sputum are problems.

Pacatal had a beneficial effect on patients suffering from energy increase due to high dosages of isoniazid.

In cases of chronic pulmonary emphysema, Pacatal proved to be a safe sedative without depressing the respiration.

The conclusions of the findings of this study suggest that an investigation on a larger group of patients and a comparison of these patients with a well-controlled group in the hospital at the same time would be most contributory.

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ADDITION OF PAS to isoniazid therapy for patients with noncavitary tuberculosis does not appear to increase the incidence of sputum conversion of roentgenographic improvement. It has been noted that drug toxicity is somewhat more frequent among patients receiving both drugs. Isoniazid administered alone does not appear to favor the emergence of isoniazid-resistant strains of tubercle bacilli.

Approximately 30 to 40 per cent of patients with slight disease demonstrate roentgenographic improvement. Among patients with moderate disease, improvement usually appears during the first four months of treatment with both isoniazid and PAS, but, from five to eight months, the trend is reversed in favor of those receiving the single drug.

Within four months, sputum cultures become negative for tubercle bacilli in more than half the patients, regardless of the stage of the disease. However, from five to eight months, infection proceeds more rapidly with combined therapy than in slight cases and with isoniazid alone in moderate cases. In nearly all the patients studied, infection cleared within eight months of therapy.

Isoniazid was given to 134 patients in dosage of 100 mg. three times a day, and 4 gm. PAS was added to each dose of isoniazid for an additional 148 subjects. Resistant strains developed in only 6 of these patients, 3 in each of the therapeutic groups.

No toxic symptoms were seen in patients treated with isoniazid alone. Of those given combined therapy, 20 had toxic reactions during the first four months and 4 reacted during the five-to-eight-month period.

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Psychosomatic Aspects of Heart Disease

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PSYCHOSOMATIC PROBLEMS are easier to prevent than to treat. The physician who first sees the patient and recognizes the problem early may prevent it from becoming psychologically fixed. This paper will describe some of the more common manifestations in relation to the heart and methods of treatment.

The term psychosomatic, as employed here, refers to the pathologic relationships that may arise between the emotions and the physiologic functioning of the heart and adjacent organs.

In our culture, heart disease carries as much dread as cancer. More than any other disease, it connotes sudden death. It further suggests the possibility of chronic invalidism, loss of employability, loss of security, and inability to care for one's family. Unfortunately, the patient is unable to separate serious from mild forms of heart disease. Even the most sophisticated find it difficult to conceive of a functional murmur as being other than a serious threat to life.

CLASSIFICATION

One may study this problem by placing the patients in three broad groups as they present themselves to the physician: those with chest pain of noncardiac origin, those with functional cardiac disorders, and those with organic heart disease in whom the emotions play some role in symptom formation.

The first group includes acute strain of muscles of the chest wall due to lifting or coughing, pleurisy, pleurodynia, contusions, neuralgia, neuritis, and chronic intercostal or pectoral muscle spasm due to nervous tension.

The second group includes functional murmurs; tachycardia and palpitation; arrhythmias; and cardiac neuroses variously designated as

"irritable heart," "effort syndrome," and "neuro-circulatory asthenia."

The third group consists of organic lesions with associated emotional concomitants, such as coronary disease and the fear that accompanies angina; postinfarction syndrome, including depression; delayed recovery or denial of illness; hypertension with its fear of a stroke; cardiac decompensation and the ancient fears associated with dropsy; rheumatic and congenital heart disease with their implications for progressive disease and shortened life; and acute pulmonary edema with its natural fear of suffocation.

COMMON PSYCHOSOMATIC SYMPTOMS

The manifestations commonly presented may be pain, respiratory and cardiac rhythm disturbances, palpitation, and extracardiac symptoms.

Pseudoanginal pain is more often in the left pectoral region, the patient usually describing the location by placing his hand over the nipple area. Occasionally, in women, the pain may be under the left breast.¹ The pain is frequently described as extending into the left axilla and, less often, down the left arm. While the aforementioned are the common areas, any portion of the thorax may be the source of pain ascribed to the heart by anxious patients; even the abdomen may be implicated. Differentiation from true angina may be made on several bases. Vagueness and atypical history are common in psychosomatic disturbances. Freedom from pain when busy and exacerbation when at rest are usual signs.

Respiratory disturbances may best be labeled pseudodyspnea. The common manifestations are frequent sighing, a feeling of heaviness in the chest, and hyperventilation. It is well to remember that the latter may lead to faintness, tachycardia, and paresthesias of a frightening degree.

Giddiness is a common complaint in this group of patients.² It most often occurs on sudden

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changes of position. Almost invariably a careful history reveals anxiety and other symptoms of emotional disorders, such as weakness, fatigue, insomnia, sweaty palms, and involvement of other organs.

IATROGENIC DISEASE

No discussion of psychosomatic disorders would be complete without consideration of the term, "iatrogenic disease." This term, meaning "doctor-induced" disease will, I am sure, haunt us forever. In the area of emotional disturbances, it refers to the fears and anxieties engendered in the patient by thoughtless statements or mutterings of the physician as he examines his patient. Perhaps we frowned as we carefully and repeatedly examined the chest much longer than anyone else had ever done. Without explanation, how is the patient to know that extraneous sounds from fans, footsteps, coughs, conversation, or a hairy chest precluded an adequate examination? or that thinking about our forthcoming golf match had distracted us and required repeated listening? or that our frown represented a transient gas pain or heartburn? The classic, noncommittal "Hmmm" that we emit to stall for time or to appear profound has never improved the well-being of a single patient. To him this signifies that his doctor is puzzled, confused, or has discovered a most interesting finding that may titillate the physician while destroying the patient.

Is there one among us who has never said, "It is not serious," (meaning, "Never fear, I know what this is."), "but you had better take it easy!" (meaning, "I am really not sure and thus wish to protect myself from recriminations.")? How does one "take it easy?" Does he walk instead of run? avoid stairs? avoid coitus? go to bed early? work half-days? take a vacation?

Have we never attempted to bolster our own ego by saying, "You came to me just in time!" "You nearly died!" "It was the worst case of its kind I have ever seen!"

What have we done to the youth with rheumatic fever who we banned from sports for all time? How many cardiac cripples exist because we were uncertain whether the murmur was functional or not? How many neuroses have we helped to create with the electrocardiogram?²³ Must every T wave and every S-T segment abnormality be mentioned to the patient, causing doubts and fears of serious heart disease?

DIAGNOSIS

Diagnosis in medicine must always be based on a careful history and physical examination. As

part of the latter, one must not forget close observation of the patient's behavior, such as the way he walks into the office, responds to greetings, sits down, speaks, grimaces, and points. These observations give much vital information which most of us receive intuitively and incorporate into our total evaluation of the patient. I should like to make a plea for more conscious scrutiny of the nuances of behavior manifested by our patients in order to aid in more definitive diagnosis.

Perhaps the most common fault in the diagnosis of psychosomatic problems is the pernicious habit of making diagnoses by exclusion. In such instances, one states, in effect, to the patient, "After doing all of these tests and examinations, I find nothing organically wrong; therefore, your trouble must all be in your head!" The fallacy of this concept of diagnosis by exclusion lies in the fact that one series of negative tests leads to another series and yet another as one winds his way painfully and expensively along the "organic" trail.

A corollary of this method is the "either/or" concept that insists the diagnosis must either be organic or functional in its entirety. In reality, one should attempt to ascertain how much of each factor may be present. A positive diagnosis can and must be made. This is just as possible as is a positive diagnosis of organic disease.

The first step in diagnosis involves a detailed history. This should include some details of family history in addition to the usual questions relating to the presence or absence of hereditary disease. It should include significant events, such as births of siblings, deaths, illnesses of close relatives, financial problems, divorces, separations, religious experiences, schooling, close relationships with teachers and others, and the emotional reactions of the patient to these experiences.

Of great import is the time relationship between the onset of symptoms and important events in the life of the patient. A father's death from myocardial infarction witnessed by the patient may have triggered the onset of chest pain. Without careful questioning, this fact might never have come to light. For one in middle age, the death of a friend may prompt a visit to his physician "for a little checkup and a cardiogram."

A close scrutiny of current interpersonal relationships is most valuable, for example, between husband and wife, parents and child, or employer and employee.

Vagueness of complaints is characteristic of psychosomatic disorders. Chest pain that occurs

more often at rest, shortness of breath that is pseudodyspneic, pain that is inframammary¹ or pectoral rather than retrosternal, and involvement of other organs with symptoms suggestive of emotional disorders all point to a psychosomatic disturbance.

I have found Dreikurs²⁴ queries of value in distinguishing organic from functional complaints. He asks, "What would be different in your life if you were well?" or, "What would happen if you could get a pill that would take away all your symptoms and make you completely well?" The neurotic patient who has found his symptoms providing many secondary gains will reveal this in his answers.

Finally, in one's diagnostic evaluation, a statement by the patient relative to the meaning to him of his symptoms must always be included. It has never been clear to me why so many physicians resent the patient's presenting his own diagnosis. We need not agree, but it can be so revealing. How often have we heard our patient shyly confess that his neighbor had just succumbed with similar symptoms.

TREATMENT

Even as diagnosis begins with a careful history and physical examination, so does treatment of the patient with psychosomatic heart disease. Whatever our skill or prestige, the patient will never consider us an expert in his particular case until we have performed these vital preliminaries.

During the examination, we have an excellent opportunity to impart positive suggestions to our patient who awaits breathlessly, with perspiration oozing from his axillae, for our pronouncements as to his fate. Shall we, then, take his blood pressure and say nothing or grunt, "Blood pressure's o.k.?" I say "No!" Here, if findings warrant, is the place to state positively and cheerfully such words as "Your blood pressure is perfect!" or, "Just like a 20-year-old!"

On examining the fundi and finding them normal, or nearly so, why not state, "Your vessels are remarkably free from sclerosis."

On listening to the lungs, why not state, "Your lungs are splendid—clear as a bell!"

And now the heart. A quick listen may satisfy the physician, but he should make the examination suffice for the patient's needs. What to say after auscultation of just another normal-sounding heart? Could we not take our cue from the family doctor whose cheery comment was proudly related to us by a patient, "My doctor said, 'That's the best heart I have ever listened to!'"

Deciding how much examination to carry out

is an important part of treatment. It may be better in some instances to advise the patient that an electrocardiogram is not necessary. In other words, the doctor is announcing, "After examining you, I feel certain that I know what your trouble is, and this test would provide no information of value."

To one patient, this might prove most reassuring. On the other hand, if it appears that the patient will not be satisfied until he has had an electrocardiogram made, why not perform it, even if the physician sees no need for it?

After completing the examination, one must decide on a way to inform the patient of the findings.

In the first category of patients, those with chest pain of noncardiac origin, it is relatively easy to reassure with buoyant optimism that the trouble is not related to the heart. Of course, a rational explanation of the cause must be offered.

In the second group, those with functional murmurs or a full-blown cardiac neurosis, the problem becomes far more difficult. A functional murmur in a stable person, who, in error, has concluded that he has heart disease, can usually be explained to him and the anxieties dispelled. In the patient with a cardiac neurosis, as in any neurosis, the problem may range from difficult to impossible to resolve. Here, the neurotic needs of the patient must be evaluated. If the secondary gains to be reaped are too great, we may be unable to encourage the patient to give up his symptoms. If the dependent wife can only gain her husband's sympathy and attention with her cardiac symptoms, why should she surrender them to please the doctor? It is well to remember that not every patient who pleads for relief of symptoms truly wishes to give them up.

If the condition is relatively fresh—say a few months in duration—one may strike out boldly, take a stand, and state definitely, "You have a normal heart! Your trouble is due to nervousness!" This may then be interpreted to the patient in some detail.

On the other hand, if the patient with a cardiac neurosis has seen many doctors over a period of years, one must proceed cautiously and gently. In such instances, one learns much from the patient's response to questions during the examination. If he shows great irritation to questions oriented toward uncovering emotions, we would be well advised to tread softly. A blunt statement to the effect that, "There is nothing wrong with your heart! It is all in your head!" could merely drive the patient to still another doctor. Such a declaration would imply

to the patient that his present doctor believes him a fool, malingerer, or worse. What can he tell his family? Were he to accept the physician's diagnosis, what would replace the gains his illness had brought him in the way of attention and sympathy or escape from unpleasant duties or responsibilities? The rule should be that we never take away a crutch unless we replace it with something equally good or better. In such an instance one might state with conviction, "I am happy to be able to tell you that you have a normal heart!" This should be followed by a detailed analysis of all the arguments in the history, physical examination, and laboratory studies that support his contention.

Then we must approach the problem of defining the role that his emotions play in creating the illness. Here we can profitably draw upon our past experience with similar cases and recite several examples with an optimistic outcome.

It is almost always necessary to give such patients a new medication, if only an inexpensive sedative, such as phenobarbital. This is particularly true if one has the problem of weaning the patient away from other drugs, such as digitalis or quinidine.

If, in the course of studying the patient, it is felt that psychotherapy is indicated and acceptable to the patient, referral to a psychiatrist should be discussed. This is an art in itself. It is at this point that the physician's feelings are as important as the patient's in determining a successful outcome.⁵ The referral shall probably fail at its inception if the physician is basically hostile to either psychiatry or to patients with neurotic complaints.

Even if the referral is good and the patient chooses to go to a psychiatrist, his physician should not renounce interest in the case. Rather, he should continue to follow as closely as necessary to provide the *reassurance* so badly needed until that need disappears. "Reassurance" is italicized to remind us that it comprises our most valuable commodity. Our patient is more interested in prognosis than diagnosis. "Is it serious, doctor?" "Will I live a normal life span?" "Will I be disabled?" These are the important questions he wishes answered. It may be necessary to reassure repeatedly for many months. The physician must not show annoyance when his patient's quest for assurance begins to sound like, "Doctor, do you really know what you are doing?" Accept his need and gratify it.

What should be done when the patient who obviously needs intensive psychotherapy rejects it? The physician must handle the problem as best he can. Remember, reassurance is our

greatest offering. Use it freely, but, of course, base it upon the facts. Allow the patient to come in as often as his needs dictate. On each visit gently question him in regard to the emotional aspects of his life and the relationship between his symptoms and his daily experiences. If he withdraws or becomes aggravated with such questions, change the subject—only to bring it up again at the next visit. I have seen gratifying results from so simple a procedure after two years of persistence. Then, the patient may come in saying, as though it were an original discovery, "Doctor, I finally found out why I get that palpitation. It always comes when I am doing twice as much work as others at the office, while they get credit for it."

The physician finds his greatest challenge in the patient with organic heart disease and a functional concomitant disorder. In such instances, it is often the needs of the physician that determine the course of treatment.⁵ If his need to be always correct is too great, he may be unable to take a firm stand and state with conviction, "Yes, you have had a myocardial infarction, but your present symptoms are psychosomatic. They represent your emotional reaction to your heart attack. Knowing this, I urge you to return to work and attempt to ignore your pain. I predict that it will gradually disappear as you regain confidence and become active once more."

Certainly, every physician knows that his patient must die some day. Why must we fear recriminations of relatives if he dies at work or while exercising rather than while "taking it easy?"

In dealing with such problems, there is no place for weasel words. Even though there may be some uncertainty in the physician's mind, he owes it to his patient to take a firm stand and hide his doubts. Of course, he will err on occasion. The man who fears to err does not belong in medicine. It is far better to be mistaken occasionally while giving patients the support, encouragement, and sympathy they need than to blunder with every patient along a noncommittal path that leaves him frightened and alone.

Is it not strange that our training has taught us that to overlook an occasional organic diagnosis is the greatest of crimes, whereas to disregard the prominent emotional factors that prevail in more than 50 per cent of all our patients is no crime at all?

SUMMARY

Careful and prompt handling of patients with chest complaints may prevent the formation of

disabling psychosomatic disorders. Chest symptoms always bring fear of heart disease. This, in turn, creates great anxiety concerning sudden death.

Patients may be placed in three broad groups: those with noncardiac chest pain, those with functional cardiac disorders, and those with organic heart disease with an emotional overlay.

Common psychosomatic manifestations are described.

The role of the physician in creating psychosomatic problems is studied.

Diagnostic points and pitfalls include careful observation of the patient's behavior and responses, avoidance of diagnosis by exclusion, and correlation of the onset of symptoms with important events in the life of the patient.

Treatment, like diagnosis, begins with a careful history and physical examination. Employ the examination as a time to reassure the pa-

tient in vivid terms as to the splendid state of his various vital organs. Evaluate the patient's needs and plan treatment accordingly. Accept limited goals if circumstances so dictate. Psychiatric referral, when indicated, should be done in a positive manner. The physician should come to know himself. He should be able to take a firm stand in patients with organic heart disease and not fear to "go out on a limb." He should discard the idea that it is better for patients to die while "taking it easy" than to die while pursuing as normal a life as possible.

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CORONARY HEART disease should no longer be considered as rendering a patient ineligible for life insurance. Life expectancy of selected groups has been shown to be far greater than was formerly supposed. Insurance coverage is usually limited to those persons not expected to exceed a long-term mortality ratio of 500 per cent, or 5 times as many deaths as would occur in the general population. The mortality ratio during the first two years following a coronary attack is 600 to 700 per cent. However, the ratio improves to 400 per cent in the third to fifth years, 300 per cent in the sixth to tenth years, and 150 per cent in the eleventh year.

As a rule, 3 of 5 persons surviving acute myocardial infarction live another five years, and 1 of 3 lives another ten years. Milder attacks, more complete recovery, and older age improve the long-term outlook. However, when disorders predisposing to progression of coronary disease, such as diabetes, obesity, hypertension, and elevated serum cholesterol, are associated, life expectancy is considerably shortened.

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Return to Employment for Cardiac Patients

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PRODUCTIVE WORK after onset of clinical heart disease has become so well accepted in the past decade that a review of some rehabilitation aspects is imperative. The problem is an increasingly dominant one, demanding the interest of private practitioners. While medical management has improved, social and employment problems may still retard the rehabilitation process. This is especially true in a period of surplus labor when many business and industrial firms provide health and accident, life, and other insurance protections as fringe benefits of employment. We also know that cardiac patients who cannot be rehabilitated for employment will, in most instances, become dependent on some form of social subsidy. Increasing concern with the social problem of disability and rehabilitation is felt not only by various social agencies but also by the private physician in behalf of his patient. It is therefore expedient for the physician to acquaint himself with all available resources that will help him return his patient to the greatest personal, social, and economic level of which he is capable.

Studies¹ have shown that about three-fourths of patients who have first heart attacks return to either full or part-time employment. Yet we know there is a large residual number who, because of fear, uncertainty, or lack of problem-solving help or information are lost as productive members of society. The family physician can and should, in most cases, give the guidance, encouragement, and assistance his patient needs to arrive at realistic conclusions, not only about his physical disability but also its effect upon the social and economic aspects of life. It has been estimated² that about 20 per cent of the cases will require referral to a team approach if the rehabilitation goal is to be realized. These are patients whose unusual problems of emotional,

family, and vocational adjustment require the skills of professional workers in these areas.

Lest we find comfort in the substantial number of cardiac patients who do return to employment, let us remember that business and industry are having an increasingly difficult problem with this disability group. The recent study in Minnesota by Mathy³ indicates something of a trend that has been observed in other parts of the country as well: a small but expensive percentage of heart attacks are now being charged against the cost of employment by way of workmen's compensation. The relatively high cost of these compensation claims causes many industries to be increasingly reticent to hire or re-employ cardiac patients. Mathy's study also points out that employer reluctance is heightened by the cost of certain employee benefits such as health and accident, life insurance, and medical policies paid by the employer. In a surplus labor market, industry generally follows a policy of restrictive hiring.

Most business and industrial firms will allow their employee to return to his job, or, if necessary, a modification of it, after a cardiac attack. This is an important point to remember when counseling your patient. If reemployment by the same firm is not possible, the chances of new employment frequently are poor for the reasons cited and because of age barriers. The physician has a responsibility here; he is frequently the only professional person who can help his patient with the closely related, nonmedical aspects of rehabilitation. The idea of rehabilitation is beautifully simple: "What does the patient need to return to his greatest level of social and economic independence?"

THE PHYSICIAN AS COUNSELOR

Medical counseling is a central aspect in most of what has been written on rehabilitation of the cardiac patient. It is well summarized by Phillip R. Lee² who says, "The single most important step in the rehabilitation of the cardiac is the physician's unhurried discussion with the patient about the nature of his disease, its treatment and prognosis and how he can best arrange his life to fit the circumstances."

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An optimistic, positive approach is usually possible—and advisable. The family also will require this approach, because personal and family reaction to the problem may be more disabling than the actual physical residuals. With mature and perceptive patients, this process may be limited to 1 or 2 interviews, possibly using supplementary reading materials; others may require several discussions. In some refractory cases, referral to a counseling agency may be indicated. Remember, there is an important difference between “telling the patient” and helping him understand. Other problems frequently encountered are readjustment of life-style, interspersing of physical and emotional strain in employment, and, in some cases, occupational downgrading.

Emotional reaction to the disability may be one of the important related problems with which the physician must deal. Reactions to a heart attack are generally an aggravation of basic personality structure. Although the patient may reach a point of acute anxiety, it is seldom of such a nature that psychiatric help is needed. The attending physician who accepts his role as the patient's counselor can and should handle these feelings and fears. He has worked through acute illness with the patient and has a good relationship with him. He is the object of the patient's hope for life and recovery. He also has considerable knowledge of the patient's habits, work history, and family situation.

A recent study by the federal government⁴ revealed that most physicians would like more information about resources and agencies that could help them in the rehabilitation of their patients. Referral resources are varied in extent and quality, depending on the locality and personalities involved.

A number of referral resources are worthy of mention here. A cardiac work classification unit has recently been established in Minneapolis. It is similar to units now in operation in Cleveland, Detroit, New York, and Chicago. It is sponsored by the Minnesota Heart Association and is under medical leadership. A cardiologist, social worker, vocational counselor, and other specialists as needed evaluate the physical, social, and vocational potential of the patient. After an evaluation by each specialist, a conference is held to determine what can be done and how to proceed in returning the patient to employment. The referring physician is a member of the team and is requested to attend this conference. The classification unit does not treat the patient. It objectively measures work potentials and matches these potentials with employ-

ment possibilities. This unit is a service to physicians and in no way competes with them. Similar projects in other cities have had excellent success in getting cardiac patients back to appropriate employment. All Minnesota residents are eligible on the referral of their own family physicians. Financial arrangements are on a liberal ability-to-pay basis, with deficits being made up by the Minnesota Heart Association. Further information may be obtained from the Cardiac Work Evaluation Unit, care of Kenny Institute, 1800 Chicago Ave., Minneapolis.

Another resource available in all states, to rural and urban areas alike, is the Division of Vocational Rehabilitation. Financed jointly by the state and federal governments, its chief function is to prevent economic dependence by offering vocational guidance, training, job placement, and related services.

Trained vocational counselors cover all areas of each state, working closely with state employment services, county welfare boards, schools, and numerous public and private agencies. These agencies may be used by the vocational counselor in the vocational rehabilitation of the patient. Evidence of a disability that constitutes an employment handicap is established by medical examination. These data are reviewed and interpreted to the counselor by a consulting physician. The counselor then proceeds with services that will help the patient return to employment. The active cooperation of the referring physician and the vocational counselor can be most important to ultimate success.

For patients too disabled for substantial gainful employment, the local security office may offer help in applying for social security disability pension⁵ to those 50 years of age and over or for freezing of earning records to those under 50.

Other resources available include visiting nurse service, family counseling agencies, welfare boards, ministers (with a flare for counseling), local sheltered workshops when available, and volunteer service bureaus (for those who need “something to do” while building tolerance for return to work or for those who cannot return to work). Most larger communities also have recreational programs that may be helpful. Some cities, including St. Paul and Minneapolis, have “coronary clubs,” which can often help patients make good adjustments.

SUMMARY

1. Rehabilitation is a point of view; it involves optimum physical, social, and economic recovery of the patient.
2. The physician must be a counselor and co-

ordinator to effect rehabilitation. He must help the patient understand his illness and direct the patient to community resources he may need.

3. Reemployment is usually more feasible than new employment. Most employers will rehire cardiac patients; however, disability and age factors are major barriers to new employment.

4. Referral means "making sure the patient actually receives the service requested of the referral agency."

5. Community agencies can be helpful if used wisely by the physician.

PAMPHLETS AND BOOKS FOR USE BY CARDIAC PATIENTS

NEEDLEN, ROBERT J., M.D., and EDITH M. STONEY: *A Primer for Coronary Patients*. New York City: Appleton-Century-Crofts, Inc.

IOWA HEART ASSOCIATION: *A Safe Work Load for Farmers with Heart Disease*. 2100 Grand Avenue, Des Moines 12, Iowa.
AMERICAN HEART ASSOCIATION: *Heart Disease Caused by Coronary Atherosclerosis; These Hands are Able; Returning Cardiacs to Work*. 44 East 23rd Street, New York City 10, New York.
CHICAGO ASSOCIATION OF COMMERCE AND INDUSTRY and CHICAGO HEART ASSOCIATION: *Problems of Retirement in Industry*. 1 North La Salle Street, New York City 28, New York.

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VENTRICULAR FIBRILLATION may be induced by surgical treatment of aortic insufficiency by closed or indirect methods or by open technics utilizing extracorporeal circulation. Cross-clamping the aorta increases the cardiac burden of aortic insufficiency, and a cardiopulmonary bypass puts great stress on the left ventricle.

Experimentation involving dogs has suggested that the hemodynamic burden on the heart may be relieved by redistribution of blood from the left ventricle. A bypass is created from the left atrium to the distal aorta, and the descending aorta is simultaneously occluded with onset of extracorporeal pumping. If the caval blood is diverted into an oxygenator, only a small quantity of blood need be bypassed from the left atrium to the distal aorta to relieve left ventricular end-diastolic hypertension. Continuous measurement of systolic and diastolic pressures within the ventricle must be employed to regulate the amount of blood entering and leaving the ventricle. Excessive diversion of blood from the atrium and, consequently, from the ventricle results in inadequate flow to the coronary and cerebral vessels.

A more objective criterion than dilation is left ventricle pressure, which is easily monitored. Pressure determinations are particularly valuable when the left ventricle is not well exposed. Cross-clamping of the descending aorta combined with perfusion of blood from the inferior vena cava to the distal aorta lowers the pressure within the cava and blocks blood flow through the azygos system into the central and cephalad reservoir. However, since redistribution of blood from the cephalad to the caudad portions of the body cannot be precisely regulated by indirect means, cardiac dilation and diastolic hypertension are not prevented by simple bypass of blood from the inferior vena cava. Although diastolic hypertension is relieved by ventriculotomy, perfused blood may be lost.

R. H. CLAUSS, R. W. BUBEN, and W. A. ALTMEHLER: Selective redistribution of blood in surgical treatment of aortic insufficiency. *Ann. Surg.* 150:586-594, 1959.

Clinical Significance of Notched T Waves

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THE T WAVE is one of the least specific but, for the same reason, also one of the most sensitive electrocardiographic variables. Levine¹ lists 67 conditions which may produce abnormal T waves, among them 18 infectious diseases. In view of the general importance of abnormal T-wave changes in clinical electrocardiography, minor T-wave changes have repeatedly attracted attention. Kiessling and associates² found in longitudinal studies a significantly increased mortality in life insurance population with small (borderline) T-wave changes, such as flat T waves in V_5 and V_6 as compared with life expectancy of the healthy population of the same age.

This study is concerned with notched, positive T waves in serial observations. Although notching of the T wave is a relatively minor irregularity, we have been observing transient notching of the T wave for several years, and the 16 cases selected are representative of a larger number of patients.

MATERIAL AND METHOD

Sixteen hospitalized patients with notched T waves in any of the 12 conventional leads were chosen for study. Except for the notched T wave in one or more of the serial tracings, the electrocardiogram was normal. The 16 patients were distributed in two groups.

Group 1 was comprised of 9 patients (table 1) with miscellaneous types of clinical disease except angina pectoris, including 3 with infectious disease (cases 3, 4, and 6), 1 case with diabetes mellitus, arterial hypertension, and hyperuricemia (case 7), 1 with anemia due to iron deficiency (case 9), and 1 with mixed tumor of the parotid with benign prostatic hypertrophy (case 2).

Group 2 consisted of 7 patients (table 2) with typical angina pectoris, 2 of them compli-

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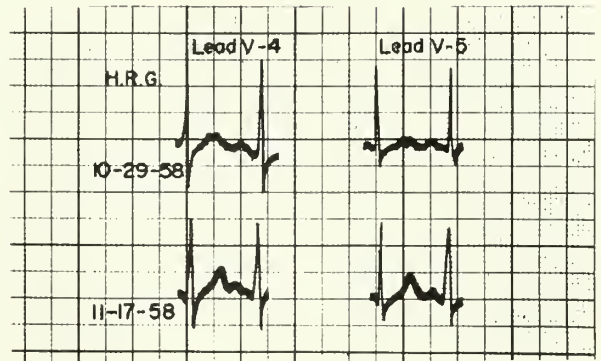


Fig. 1. Case 2, group 1. Notching of positive T wave disappears with clinical improvement.

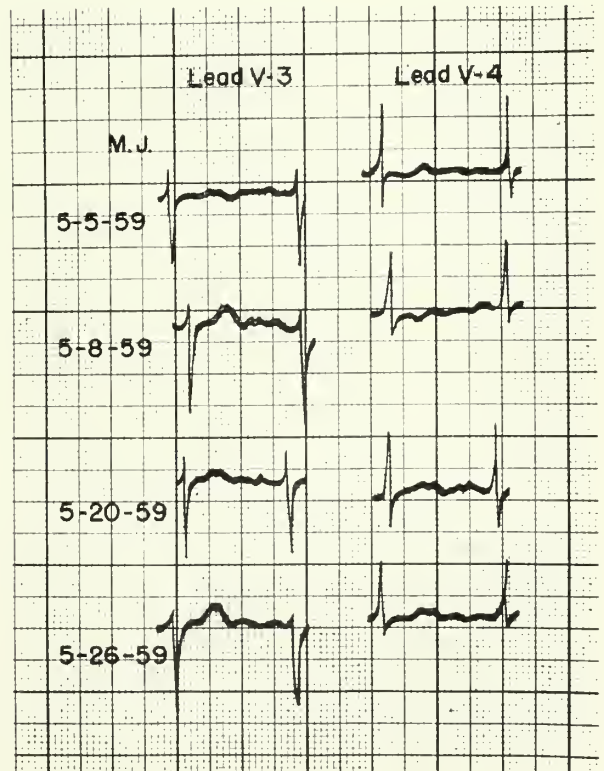


Fig. 2. Case 9, group 1. Notching of a positive T wave follows a diphasic T wave on 5/8/59 in lead V_4 . In lead V_3 , the T wave is notched in all 4 observations. T wave was normal in other leads.

TABLE 1
SERIAL TRACINGS OF NOTCHED POSITIVE T WAVES IN PRECORDIAL LEADS
GROUP 1. MISCELLANEOUS, EXCEPT CORONARY INSUFFICIENCY

Case and Diagnoses	Sex	Age	Blood pressure	Date of EKG	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
1. Acute benign idiopathic pericarditis	F	44	110/20	11-20-58 11-24-58 11-28-58	+	+	+	+	+	+
2. Mixed parotid tumor, benign prostatic hypertrophy	M	65	150/90	10-29-58 11- 3-58 11-17-58	+	+	N	N	+	+
3. Ulcerative nose and throat (staph.), diabetes mellitus	F	30	125/76	10-29-58 11- 3-58	N	N	+	+	N	+
4. Encephalitis, myocarditis, convulsive disorders	F	28	98/66	12-28-58 1-31-58 1- 5-59 1-10-59	+	+	N	N	N	N
5. Hypercholesterolemia, nervousness	F	56	130/65	1-13-58 1- 5-59 7- 6-59	+	+	+	+	+	+
6. Pyelitis, pyelonephritis, toxic myocarditis, probable virus pneumonia or mononucleosis	F	19	104/65	1-27-59 1-30-59 2- 2-59 3- 2-59	+	+	+	+	+	+
7. Diabetes, hyperuricemia, abnormal liver function, nervousness	M	41		12-17-58 12-22-58	+	+	+	N	+	+
8. Cerebral concussion, traumatic pleural fluid, fractured right rib, renal and liver concussion, right basilar atelectasis	M	45	95/20	5-13-59 5-17-59 5-18-59	+	+	N	N	N	N
9. Anemia	F	21	164/56	5- 5-59 5- 8-59 5-10-59 5-20-59 5-26-59	+	N	+	s+	s+	Hb 5.6
					+	N	+	s+	s+	Hb 5.5
					+	s+	+	s+	s+	Hb 5.4
					+	N	N	+	+	Hb 6.8
					+	N	N	+	+	Hb 7.7

F, Flat T; N, Notched T; +, Positive; -, Negative; s, Small; +-, ++, Diphasic

ected by acute gallbladder disease (cases 3 and 6), 1 with a calcified aortic stenosis and coarctation of the aorta (case 5), and 1 with a patch of pneumonia in the roentgenogram (case 7).

The female sex predominated in the first group and the male sex in the second group. Ages in the first group ranged from 19 to 71 years and, in the second group, from 52 to 71 years. The presentation is limited to the precordial leads; notching of the T wave in the limb leads was less common and not present in these patients.

RESULTS AND COMMENT

Group 1, table 1: In cases 2, 7, and 8, the notched positive T waves changed to smooth, positive T waves as the patient improved (figure 1). In cases 4, 5, and 9, notched T waves are a residue of preceding abnormalities as shown by negative or diphasic T waves (figure 2). In cases 6 and 9, notching of positive T waves preceded development of inverted or diphasic T waves.

Group 2, table 2: In cases 1 and 5, the notching changed to positive, smooth T waves. In cases 2 and 3, it preceded flat, diphasic or inverted T waves and followed previous diphasic

or inverted T waves. In case 4, the notching had disappeared in the resting electrocardiogram before the meal test³ but reappeared after the meal in the same leads. In case 6, the notched T in lead V₄ changed to diphasic and notching appeared in the positive T wave of lead V₅ after the meal test. The next day, the electrocardiogram showed notching of the T waves from leads V₄ to V₆.

In general, the notching of T waves was more common in leads V₁, V₂, V₃, and V₆. Only 5 cases had a notched T wave in leads V₁ or V₂ (table 1, cases 3 to 6; table 2, case 7).

A notched, positive T wave as an isolated finding is comparatively rare. It was present only in about 1 per cent of the electrocardiograms during the time these data were collected. A notched T wave at the edges of a zone with inverted or diphasic T wave is quite common but has little, if any, additional diagnostic significance and was not included in this study.

A notched T wave may precede or follow an abnormal diphasic or inverted T wave. From theoretic considerations, a notched, positive T wave may be expected to precede a diphasic T wave. Therefore, a notched T wave may be the

TABLE 2
NOTCHED T WAVES
GROUP 2. PATIENTS WITH ANGINA PECTORIS

Case and Diagnoses	Sex	Age	Blood pressure	Date of EKG	V ₁	V ₂	V	V ₄	V ₅	V ₆
1.	M	57	120/75	8-16-58	+	+	N	N	+	+
				8-19-58	+	+	+	N	+	+
				8-24-58	+	+	+	+	+	+
				8-28-58	+	+	+	+	+	+
2. Paroxysmal auricular tachycardia	F	71	150/90	11-4-58		++		F	F	F
				11-5-58		++	++	++	++	++
				11-7-58		++	++	++	++	++
				11-11-58		++	++	N	N	+
				11-14-58		N	N	N	N	+
				11-18-58		N	N	+	+	+
				11-21-58		N	N	+	+	+
				11-25-58	F	++	+	+	+	+
3. Acute gallbladder, anxiety	F	60	100/60	12-30-58	++	++	++		F	+
				12-31-58	++	+	+	N	N	+
				6-1-59	++	++	+	F	F	F
4.	M	61	140/80	5-13-58	+	+	+	N	N	N
				5-18-58	+	+	+	+	+	+
					+	+	+	N	N	N before meal
										N after meal
5. Calcified aortic stenosis, pseudocoarctation of aorta	M	52	120/90	4-17-59	+	+	+	N	s+	+
				5-17-59	+	+	+	N	+	+
6. Gallbladder disease	M	58	130/80	5-16-59	+	+	+	+	+	+
				5-17-59	+	+	+	+	+	+
				5-18-59	+	+	+	+	+	+
				5-19-59	+	+	+	sN	s+	s+
					+	+	+	+	N	+
				5-20-59	+	+	+	N	N	N before meal
										N after meal
7.	F	59	170/110	4-4-59	++	+	+	+	+	+
				4-30-59	N	+	+	+	+	+
				5-9-59	+	+	+	+	+	+
				5-13-59	+	+	+	N	N	N

F, Flat T; N, Notched T; +, Positive; -, Negative; s, Small; ++, --, Diphasic

first sign of developing myocardial involvement or the last residue of preceding involvement. Occasionally, the involvement may not be severe enough to produce inverted T waves, and the notched T wave then is the only transient sign of electrocardiographically mild and, usually, clinically silent involvement (group 1, cases 3 and 7). However, a transient, notched T wave exceeds the limits of normal variability when observed within a period of several weeks or months.⁴

Notched T waves obviously are not specific for any clinical condition, as shown by the variety of pathology in group 1. Infectious diseases were relatively frequent. Most of the conditions listed in group 1 are known to produce inverted T waves occasionally, and, of course, inverted T waves may be expected to develop in patients in group 2 with the progress of coronary artery disease. The fact that a positive, notched T wave is relatively rare may be due to the rapid development of abnormal T waves in most types of myocardial involvement. The relatively high figure of about 80 per cent abnormal electrocardiograms in the material surveyed may be of importance in this respect. It is possible that the

incidence of notched T waves in ambulatory patients may be higher.

In older patients, a notched T wave may be a relatively slight electrocardiographic irregularity without clinical correlations. However, our results suggest the need of serial follow-up of patients with notched T waves as the only electrocardiographic sign.

SUMMARY

Sixteen hospitalized patients with various types of pathology are presented. Notched, positive T waves in at least one of the serial tracings preceding or following diphasic or inverted T waves was the only sign of irregularity in the electrocardiogram. Notched T waves may be the earliest or latest sign of myocardial involvement.

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Retinal Detachment

ROBERT FINK, M.D.

STATISTICALLY, retinal detachment is not a common problem. An ophthalmologist will see only 1 or possibly 2 such cases in a year's time. And yet, detachment can be one of the most perplexing problems we have to face. Such a case challenges our abilities to their fullest capacity.

Surgery requires much precision and care but will be compromised from the start unless the patient has been thoroughly studied beforehand. Diagnosis is not difficult; the problem is to find the tears or holes in the retina that caused its separation and to determine the type of treatment required to best combat the pathologic changes in the retina and vitreous fluid.

The role of the physician is an important one in these cases and requires close cooperation with the ophthalmologist. It is the physician who may have to decide whether an elderly patient is fit enough to endure two to four hours of general anesthesia and two weeks in bed with a minimum of movement. The patient may be gambling with his life against certain blindness if detachment has occurred in his only remaining eye. The physician may be called upon to administer steroids if the eye shows evidence of active inflammation, either pre- or postoperatively. And, finally, the emotional factor must be seriously considered. These persons have experienced sudden blindness and are faced with imminent major surgery. They are often understandably anxious and depressed, especially if the other eye has been treated unsuccessfully for the same condition. The long-term postoperative care of these patients is much more difficult if they are not handled with a great deal of tact and care by both the physician and ophthalmologist. The odds of success are generally

not good. The over-all rate is about 75 per cent but individually may be as low as 5 per cent in the more severe detachments. Enthusiasm is no substitute for tempered encouragement. If the eye does not do well postoperatively, the patient may have to be let down gently, sometimes over a period of weeks. Very few can stand to have their hopes abruptly dashed after the ordeal they have been through without becoming extremely depressed. You, as their family doctor, often have a more personal relationship with these patients initially, and your opinions will be sought. You should have a general idea of the condition and treatment involved. This article is designed to give you a brief synopsis of the current thinking and treatment of retinal detachment.

GENERAL COMMENTS

Retinal detachments most frequently occur in the 50 to 60 year age group. Some juveniles have congenital weakness in the retina, often symmetrically and bilaterally, but they are a minority. Clinically, only 10 per cent of detachments are the result of trauma. A blow to the head may knock loose an impending detachment, but the eye itself must be severely injured to cause a separation. This occurs either immediately by the tearing loose of the normal retinal attachments or later by the traction of an organized hemorrhage in the eye.

Roughly 50 per cent of all detachments occur in nearsighted people. The exact reason for this is as yet unknown, but one can see the predisposing degenerative changes in the retina of these myopes. Apparently, there is an obliteration of the small vessels in the peripheral retina that results in atrophy and a weakened condition.

Another 20 per cent of detachments develop within three years after cataract extraction. They are not the result of poor technique or complica-

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tions at the time of cataract surgery in most cases. Actually, most patients who have detachment had completely uneventful surgery. Persons who fall in this group are usually about fifteen years younger than those with true senile cataracts, and their lens changes are just part of a dystrophic eye condition. The prognosis is poorer in these aphakic detachments, as they are of a more complicated type and are prone to further deterioration after surgery.

In the 2 latter categories, myopes and aphakic patients with retinal detachment, the physician and the ophthalmologist must be aware that over one-third of these patients will eventually have a detachment in the other eye. This makes it more imperative to salvage the vision, for the operated eye may eventually be the better eye.

PATHOGENESIS

The posterior two-thirds of the eye is spherical and composed of 3 layers: the outer tough sclera, the middle vascular choroid, and the inner sensory retina. The cavity is filled with vitreous gel. This fluid is the most important factor in causing a retinal detachment or complicating its recovery. It is a thick, viscous fluid that contains collagen fibrils and appears to have the consistency of raw egg white.

When this vitreous body undergoes pathologic change, it tends to shrink in size. There is also an increase in the amount of connective tissue strands, and these, being elastic, tend to contract. This shrinkage of the gel and pulling of its strands puts tension on the retina, to which they are attached. A healthy retina can withstand such tension, but a retina that has been weakened in spots by areas of degeneration may tear. These weakened areas can be observed and are usually the result of inflammation, degeneration after closure of small vessels, or trauma. Whatever the cause, it affects not only the retina but also the vitreous.

When one or more tears develop in the retina from the tugging of the vitreous adhesions, the stage is set for retinal detachment. Serous fluid, which has filled the space created by the vitreous shrinkage, seeps through the hole in the retina. With normal eye movements, this fluid tends to slosh around and force its way behind the retina. Once there, it begins to dissect the retina free from the underlying choroid, thus depriving it of its posterior blood supply. As the fluid behind the retina increases, it detaches more and more of the retina and balloons it forward. The retina that is separated ceases to function, although it is kept alive by the retinal vessels that lie on its anterior surface.

The patient initially complains of flashing lights. This is the tugging on the retina by the shrunken vitreous. Next, he may notice black floating specks or dots. A tear has developed across a small blood vessel, and he is seeing small clumps of blood that have gotten into the vitreous fluid. Then, it is just a short time before he notices the "curtain" coming across his vision, an indication that the retina has detached. If nothing is done and the retina comes off completely, the eye is blind.

EXAMINATION

When an ophthalmologist is confronted with such a case, he must first and foremost find the holes or tears in the retina. This can be most difficult if he has to look through a hazy lens or vitreous clouded with blood in order to see the retina. The search should be thorough, and the examiner must find all the tears. Sealing off of several holes in the retina and faultless surgery do little good for the patient if a hole is still left open.

Next, the nature of the detachment must be evaluated. The more extensive the detachment, the poorer the prognosis. Multiple pigmented areas in the peripheral retina suggest old healed inflammation, while ill-defined whitish patches mean an active inflammation. The extent and degree of degeneration should be noted, as these weak areas are potential sources of future trouble and must be considered at the time of surgery.

The condition of the vitreous gel and its effect on detachment are equally important to study. Traction on the retina by shrunken vitreous strands can be seen. One sees sharp ridges in the retina or puckering of it, forming starshaped folds. These elevated folds are constant and do not change in appearance, despite shifting of the fluid underneath the separated retina. The patient may be put to bed before surgery to see if the fluid beneath the retina will absorb and permit the retina to settle back again. If it remains elevated with these fixed traction folds, the prognosis is poorer. This means that the vitreous has shrunken to such an extent that it will not permit the retina to settle. The retina is being held away from the choroid by vitreous strands. A more heroic type of surgery is required here. The condition of the structural elements in the vitreous is examined. Abnormal connective tissue strands or membranes result from an inflammation or a former hemorrhage into the vitreous. White blood cells suggest an active or smoldering inflammation in the retina that can flare up from the insult to the eye by surgery. Such a complication could be disas-

trous, so the physician must be ready to use steroids in heavy doses.

TREATMENT

The purpose of treatment in retinal detachment is obvious: to seal off the tears that permitted the detachment and to get the retina back on the choroid where it belongs. If the time interval has not been too long, the retina will again function and the patient will see. Recovery of the retina is sometimes slow, and, unfortunately, if the delicate central visual area, the macula, has been detached, it very seldom fully recovers. It would be very foolish to promise the patient his former vision. It is difficult to say how long a patient can go before it is too late to help him. The sooner the surgery is done from onset of detachment, the better the prognosis. Valuable vision has been obtained even after the interval has been two years!

Retinopexy. If the detachment is relatively small with no evidence of vitreous traction and if the retina settles back during bedrest, diathermy alone is sufficient treatment. This type of operation is called retinopexy. The outer scleral coat is penetrated by an electric cautery needle to cause a small burn. This diathermy reaction will create a small scar in the underlying choroid and retina. Multiple diathermy punctures have to be placed so that these scars will surround the retina tears and seal them off. Also, they must be placed intermittently over the area detached to make the retina adherent. Before this is done, however, any fluid still under the retina must be drained, so that the retina will be in contact with the treated area. This fluid is subretinal, so a perforation is made through the sclera and choroid, permitting the fluid to escape and letting the retina settle back. In order to expose the sclera that must be treated, the surgeon must cut through the conjunctiva and temporarily detach one or more of the eye muscles. He must locate the exact area of the retinal holes on the overlying sclera by careful observation. The patient is then confined to bed after the operation for about two weeks to permit the small scars to form. Head and eye movement must be limited so that the vitreous gel does not move around and pull on the retina before it has a chance to adhere to the choroid.

Recently, it has been possible to create a thermal reaction in the retina by focusing an intense concentration of light rays on it. The observer has the advantage of seeing exactly where he wishes to place his burns. Such a method is very useful if the retina has flattened back on the choroid. If, despite bedrest, fluid remains

under the retina, this means cannot be used. Unfortunately, this is usually the case, so the visible light treatment is limited.

Scleral shortening procedures. As mentioned earlier, pathologic changes in the vitreous (shrinkage with or without increased connective tissue) play a major role in retinal detachments. The retina is not only pushed off by the fluid seeping behind it but is also held off by varying degrees of vitreous traction. If this traction is slight, retinopexy alone will hold the retina back in place, once the subretinal fluid has been drained. However, if one sees evidence of a strong vitreous pull, one must attempt to counteract it by more complex surgery, for diathermy alone will not suffice. Since the volume of the vitreous gel has decreased by shrinking, there is a disproportionate amount of space inside the eye. The problem is handled in one of the following ways.

Scleral resection operation. Simply stated, the eye is too big for the amount of vitreous it contains, so the circumference of the eye is decreased by cutting out a strip of the scleral coat. This strip is nearly full thickness and is usually removed along the equator of the globe. After it has been discarded, the free edges are drawn together and united with silk sutures. This shortens the anterior-posterior diameter, thus making the eye smaller. There is now proportionately more vitreous, and the tension on the retina is relaxed. Diathermy is employed to create the necessary adhesions between the retina and choroid. The operation is technically hazardous, and a late rupture of the wound could be disastrous to the eye.

Scleral buckling operation. The variations of this operation have the advantage of being less dangerous to the eye and of doing more to combat vitreous traction. Here, the globe is not only shortened but a ridge is folded inward. A resection similar to the procedure above is made in the sclera, except that the strip removed is not full thickness. Only about two-thirds of the sclera is removed, leaving a trough with a floor of thin sclera. After diathermy has been applied to the floor, polyethylene tubing is placed in the trough. Once the subretinal fluid has been released, the eye is very soft and the edges of the scleral trough can be united over this tubing. The polyethylene is buried within the sclera and pushes a ridge of treated tissue inward to catch the retinal tears and to counteract the vitreous tug.

Some surgeons prefer to use the resected strip of sclera to form this ridge, rather than a foreign material. They will undermine the scleral strip

on both sides, apply diathermy beneath it, and close the parallel free edges of sclera over it. In either case, the material buried in the closed trough causes an infolding or "buckling" inward of the thin floor of the trough and the underlying choroid. The eye has been shortened, but the inner volume has been further lessened by this invagination. The ridge should be placed so that it catches the area of the retina that has the holes or tears. It will seal off these tears until the diathermy scars develop and relax any traction on the retina.

Vitreous implant. Rather than shorten the globe to compensate for the vitreous, this method attempts to increase the vitreous volume directly by injecting more vitreous into the eye. Vitreous is removed from the eye of a recently deceased person and injected into the patient's eye after the subretinal fluid has been released. Here, too, the sclera must be first treated with diathermy so that the retina will stay in place. The injected vitreous forces the retina back against the choroid and makes up for the deficit by filling the cavity. Despite the size of the needle (No. 18 gauge), the newly implanted vitreous loses its normal cellular architecture by being forced through a small opening under pressure. Although this is a foreign protein, there have been surprisingly few reactions to date. However, the results do not appear to be as permanent as those obtained with the scleral buckling procedure.

These later methods of handling the more severe detachments have greatly increased the chance of success. Most of these innovations are fairly recent, and improvements in technic are made as experience with them increases. Many patients once considered inoperable are now being cured. Great strides have been made in the last ten years, but almost one-fourth of all detachment operations still end in failure. Many problems still must be solved.

CONCLUSION

Retinal detachment is one of the more important,

and very often depressing, phases of ophthalmology. Only the highlights of this very complex subject are mentioned in this article. We are called upon to make an exacting appraisal of each case and treat it to the best of our ability. Our relationship with the patient is necessarily close and often lengthy. A detachment is not cured on the operating table. It is not considered cured until the retina successfully adheres to the underlying choroid and remains in place for six months. During the first few weeks after surgery, we watch closely for unfavorable postoperative reactions: infection, further vitreous shrinking, flare-up of inflammation, intraocular hemorrhage, or fresh seepage of fluid under the retina. When one assaults a structure as delicate as the retina as well as the vascular choroid and the unpredictable vitreous gel with the necessary mechanical and thermal trauma of surgery, one must expect a certain percentage of unfavorable reactions. Nevertheless, it can be bitterly disappointing to see a successful reattachment start to go bad after several weeks of apparent recovery. Further surgery may be necessary, or, possibly, the situation is hopeless. In either case, the patient must be handled carefully. He has already been through quite an experience.

The responsibility of the physician is apparent. He must ready the patient for surgery and confinement to bed and determine the patient's ability to withstand such procedures. After surgery, he must watch for urinary retention, drug sensitivities, and stasis in the leg veins and regulate fluid and dietary intake. He must handle antibiotic therapy, bowel problems, bed sores, administration of steroids, and, oftentimes, anxiety and depression.

Finally, lest this subject sound too morose, there is very little more satisfying than to successfully treat a retinal detachment. These patients are generally young enough to be vital and alert, and to work with them is very gratifying. Their ability to accept a poor situation and the patience they exhibit are most inspiring.

OCULAR ABNORMALITIES in the cerebral palsied child impede rehabilitation and education. Blindness and subnormal vision; disturbances of ocular motility and abnormal head postures; monocular amblyopia; and visual field defects, such as homonymous hemianopsia, are frequently seen. Diagnosis and treatment are often hindered by a combination of defects or by associated congenital and developmental anomalies.

S. DIAMOND: Ocular evaluation of the cerebral palsied child. *Am. J. Ophth.* 48:721-730, 1959.



Edwin J. Simons, M.D.

HERMAN E. HILLEBOE, M.D.

Albany, New York

GENERAL PRACTICE in rural Minnesota allows the conscientious physician little time for anything but caring for patients morning, noon, and night. Rarely does a physician come along who can escape, by skill and effort, this way of life. Such a physician was Edwin J. Simons, who practiced at Swanville, Minnesota, from 1925 to 1950. He was that unusual breed of rural physician who practiced the kind of medicine he had been taught in a great medical center. Before his death in December 1958, he contributed original work to the medical literature, was a leader in organized medicine in Minnesota, participated in the sound development of public medical care programs in the state, and gave crucial leadership as executive director to the Minnesota Blue Shield program. He was a man who not only believed in preventive medicine but practiced it at a time when most physicians were almost wholly absorbed in curative medicine. His work in tuberculosis control alone would qualify him as an expert in public health. He viewed his patients as a part of a family and a community and not just as an organism presenting signs and symptoms of disease.

Dr. Simons was born June 23, 1896, in St. Paul, Minnesota. He was the son of Hiram Austin Simons and Mary Elizabeth Gottwald. Orlando Simons, the first district judge of Ramsey County, was his grandfather. He received his elementary school education in St. Paul, went to high school in Bemidji, Minnesota, and attended college at the University of North Dakota. He was graduated from the University of Minnesota Medical School in 1924 and interned at Miller and Ancker hospitals in St. Paul

and the Swedish Hospital in Minneapolis. Dr. Simons was a member of the Phi Rho Sigma medical fraternity and was elected to membership in the honorary medical fraternity, Alpha Omega Alpha.

Young Dr. Simons got his first taste of general practice during six months at Long Prairie, Minnesota, in 1924. He was closely associated with the Drs. Frederick Van Volkenburg, senior and junior, who owned a hospital at Long Prairie. Dr. Simons continued this association in 1925 when he moved to nearby Swanville, where he set up a solo practice in two rooms over the First National Bank Building. The village, a typical dairy farming community, had a population of about 500. Little Falls, 17 miles east, provided good hospital facilities. Minneapolis was 100 miles south, and expert specialists there were quickly available for consultation when needed. Closer, and to the south, was the tiny village of Upsala with less than 100 people. Dr. Simons, with his sly sense of humor, liked to tell the story of where Swanville was located. When asked by his medical colleagues at distant meetings, "Where is Swanville?" he would reply, "Why, it's located just 11 miles north of Upsala, Minnesota."

It was this farming area of roughly 900 square miles that Dr. Simons used as his field laboratory for developing a general practice that brought modern medicine to all his patients and enabled him to study the natural history of tuberculosis, cancer of the lung, and a host of other diseases.

In 1925, Dr. Simons brightened up his home and his practice by marrying Hazel Cayott, a pretty little nurse he had met at medical school. "Tiny" Simons, as she was known to her friends, was a great comfort and help to Dr. Simons, especially in those early difficult years when a young doctor forges his skill and reputation in the fires of country practice. Tiny

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was an indispensable aide during two major misfortunes that occurred in those early years. Dr. Simons' hands were overexposed to x-rays, and several of his fingers became stiffened. Although his ability to perform digital examinations was only mildly impaired, he could no longer do surgery or practice in obstetrics. He was further handicapped when he lost the sight of one eye as a result of an automobile accident. If these misfortunes had happened to a man of lesser stature, it might have marked the end of a promising medical career. But Dr. Simons, with the help of his courageous wife, rose above his disabilities and drove himself to develop other attributes that more than compensated for the ones he lost. His patients, during their periods of pain and suffering, were acutely conscious of the deep sympathy and understanding of their physician, who knew firsthand what pain and suffering does to the mind and body. These two interruptions in Dr. Simons' career made it strongly advisable for him to have another physician join him in practice. His associates were Dr. R. S. Steffens, 1926-1927, Dr. Roland Scherer, 1927-1928, and Dr. Christian Rohrer, 1928-1929. From 1929 to 1930, I had the privilege of working with him.

A day in country practice with Dr. Simons was a week's postgraduate course all rolled up into a neat little package. We began seeing patients at the office early—about 7 A.M. The farmers delivered their cream to the cooperative dairy and brought their ailing family members to the office at daybreak. They were always in a hurry to get back to the farm chores again. No matter how crowded the waiting room was, Dr. Simons always took time to write a careful history. He always insisted upon a complete physical examination of new patients. One of his outstanding traits as a physician was his meticulous attention to details. He wanted the whole picture and would not settle for less. We performed blood and urine examinations ourselves, but anything more extensive was sent to the hospital laboratory at Little Falls. In those days, very few general practitioners had a well equipped laboratory next to their examining room.

By noon, we had cleared out the waiting room, and, after lunch, we were ready to make country calls. When we were not too rushed, we made calls together, especially in the winter. Day after day, I marvelled at the consistent meticulousness of this man as he diagnosed and treated patients of all ages in their homes at all hours of the day and night. In the office, the home, or the hospital, he always insisted upon a history, physical examination, and laboratory study. His unwavering goal was to make the most accurate diagnosis possible.

Late in the afternoon, we saw our hospital patients and, on the drive home, reviewed the interesting cases of the day. There usually was a "puzzler" or two to discuss. Just as certain as the sun went down, I received a call after dinner to come up to the Simons' house on the hill to go over an article on one of the problems we had encountered during

the day. Dr. Simons had a seemingly unlimited capacity to wade through medical journals until he found the answer he was seeking. Each difficult case was a new challenge to him. He never lost his zest for trying to unravel a medical mystery. I suppose it was this self-discipline and intellectual training that made him the keen diagnostician and therapist that he became.

This, then, was the almost daily routine of Dr. Simons, the general practitioner. He was fiercely dedicated to the healing profession, regardless of financial gain or civic award. He demanded the same high standards of his consultants that he had set for himself. If he believed that his surgical confreres in neighboring cities did not have the special skills to handle a particularly difficult case, he did not hesitate to call consultants in from over 100 miles away in Minneapolis. This custom often did not sit well with the local surgeons, but it made an impressive difference in the hospital's operative mortality record.

Early in his career, he began to attend medical society meetings regularly to gain new knowledge and to exchange information with his colleagues. He took part in the committees of the medical society and, after years of service to organized medicine, became a counselor from the Seventh District and, in 1946, president of the Minnesota Medical Association.

Dr. Simons found time to participate in village affairs and served on the village council and board of education in Swanville. He was as well known to the people as the Roman Catholic priest and the Lutheran minister. Anyone in need of his aid at any hour of the day or night could depend on him, and his help was by no means limited to medical affairs. The families under his care never seemed to mind that he knew of their weaknesses as well as their strengths, because they sensed his genuine interest in them as individuals.

In spite of his daily grind, Dr. Simons somehow found time to study the literature on his more interesting cases and to publish articles about them in medical journals. He accumulated an extensive medical library and thought nothing of having boxes of books and periodicals shipped to him frequently from the libraries in Minneapolis and St. Paul. His writings included such diverse topics as angioneurotic edema, multiple diverticula of the small intestine, vitamin A destruction, rural experiences with tuberculosis, carcinoma of the lungs, and medical economics.

Because of my personal experiences with two of these subjects and Dr. Simons' approach to their elucidation, I should like to mention in some detail tuberculosis and, later, carcinoma of the lungs. No new facts were contributed and no new theories promulgated, but the experiences set forth gave a broader concept of these entities against a rural background.

In the latter part of 1929, Swanville suffered an epidemic of whooping cough. Soon thereafter, 60

cases of measles appeared in the surrounding community, occurring until early in the spring of 1930. These two mild epidemics heralded an increase in tuberculosis—19 cases to be exact—in the following nine months.

The first case of tuberculosis was in an 11-year-old girl with tuberculous meningitis. Her spinal fluid showed tubercle bacilli. Examination revealed far advanced tuberculosis of the right lung. Contact examination uncovered active tuberculosis in the father. All but 3 of the 7 remaining members of the family showed positive tuberculin tests. Of the 6 children, 3 exhibited evidence of childhood-type tuberculosis. The 11-year-old girl, it was learned, helped to deliver milk throughout the village. The father's history disclosed that three years previously, while living on a farm, the whole family had consumed raw milk from a cow that a veterinarian later certified had died of tuberculosis.

In this family epidemic, many of the problems of tuberculosis presented themselves—1 death from tuberculous meningitis, 4 cases of tuberculosis following either whooping cough or measles, and a possible relationship between bovine and human tuberculosis. This experience put us on the alert for other cases to appear. We did not have long to wait.

A 19-year-old girl, recently graduated from high school, presented symptoms of a stubborn cold and chronic fatigue. Her positive sputum report from the state laboratory introduced us to the "teenage girl" problem in tuberculosis.

Then, a second case of tuberculous meningitis insidiously appeared. In March 1930, measles developed in a 3-year-old girl and was followed by bronchopneumonia. Her convalescence was prolonged, and, by June, she had not regained her health and began to exhibit symptoms of meningeal irritation. Guinea pig inoculation confirmed the diagnosis of tuberculous meningitis, and the child died in her fourth week of illness. The family members were tuberculin tested. The mother and an 11-year-old sister were found positive; the other 2 children had no reactions. The 11-year-old showed evidence of childhood-type tuberculosis.

In the 19 cases discovered during that nine-month period, a variety of diagnostic problems presented themselves: tuberculous meningitis, pulmonary hemorrhage, tuberculous colitis and laryngitis, and pleurisy with effusion. By following a definite routine in the observation of doubtful cases, Dr. Simons came forth with several precise diagnoses that would have been a credit to an expert chest specialist.

During the period in which clinical cases were being uncovered, the rural tuberculosis problem was attacked on two other fronts. During September 1930 in Morrison County, 607 students, predominantly from the high school, received tuberculin tests. The tests were applied by Dr. E. A. Leggett of Minneapolis, sponsored, I believe, by the Minnesota Public Health Association. Positive reactors had roentgenograms taken at St. Gabriel's Hospital in Little

Falls, and these were read by Dr. J. A. Myers of Minneapolis and Dr. H. A. Burns of the state tuberculosis sanatorium at Ah-gwah-ehing. Of the total number tested, 19.7 per cent reacted positively to either 0.1 or 1 mg. of old tuberculin administered by the Mantoux method. Roentgenogram interpretation revealed 66 findings characteristic of tuberculosis. This gives some idea of the magnitude of the rural tuberculosis problem in Morrison County in 1930. The clinical follow-up studies on these prime suspects were done by the designated family physicians.

In August 1930, the state Livestock Sanitary Board arranged for the tuberculin testing of the cattle population of Morrison County, in which Swanville was located. Of 3,453 herds tested, 5.5 per cent, or 192, were infected. Among 54,493 cattle tested, 572 were reactors, 33 were suspects, and 45 were placed under "feeder" quarantine.

The job was still not completed. The families of the owners of the tuberculous cattle needed investigation, and more cases came to light. In the Swanville area, for example, 3 cases of tuberculosis were found in the family of the owner of 1 of the tuberculous cows.

During succeeding years, tuberculosis control became a regular part of the office routine of Dr. Simons. Somehow, he found time to practice this kind of preventive medicine—early detection, accurate diagnosis, prompt and adequate treatment, and isolation of active cases. He thus prevented progression of the disease in those who were afflicted and prevented the occurrence of disease among family contacts. Who said a busy general practitioner hasn't time to practice preventive medicine?

Thirty years later, in 1960, tuberculosis continues to pose a threat to the health of unsuspecting families in many parts of the United States. The general practitioner on the lookout for hidden cases can be his community's leader in tuberculosis control. All he need do is to emulate what Dr. Simons did with tuberculin tests, roentgenograms, laboratory services, detailed histories, and careful physical examinations.

Dr. Simons' experiences with tuberculosis among rural patients undoubtedly focused his attention on other chest diseases as well. In 1930, he made a diagnosis of primary carcinoma of the lung in a 19-year-old farm boy. Lung carcinoma was at that time an uncommon diagnosis in a person of that age. Again, alertness and a systematic approach to a clinical problem paid handsome dividends in professional satisfaction. Through an analysis of 1,850 such cases reported in the literature, Dr. Simons found only 11 to be 19 years of age or younger. The clinical and laboratory work-up of the case through to postmortem examination, as reported in *THE JOURNAL-LANCET* of September 1931, could hardly have been surpassed by a professor of medicine in a leading medical school.

In July 1932, he read a paper on primary carcinoma of the lung before the Society of Internal

Medicine of Minnesota. It was at this meeting that the Society awarded him its annual prize in recognition of his work in this field.

In ensuing years, Dr. Simons reviewed literature from all over the world on carcinoma of the lungs. Every few weeks, a packing case of books and periodicals arrived from the University of Minnesota library and other sources. Trips to Minneapolis were sure to include several hours of library research for additional material.

He pursued the subject so zealously and relentlessly that by 1935 he had accumulated more than enough material to write a book. In his scarce free time as a busy practitioner, he somehow managed to complete a manuscript which was published in 1937 by the Year Book Publishers, Inc., under the title of *Primary Carcinoma of the Lung*. It was a book for general practitioners and was written by one who undoubtedly knew more about the subject than any other general practitioner of his day. The results of his tremendous effort belie the myth that to make a notable contribution to medical science a physician must have at his ready disposal the laboratories, libraries, and other facilities of a large hospital or medical school. A man possessed of the intellectual curiosity and first-class medical training of Dr. Simons can contribute to medical knowledge and the well-being of his patients, whatever his surroundings.

Today, in 1960, cancer of the lung is the first cause of death from cancer among men in the United States. Yet, in 1930, this same disease was infrequently diagnosed, especially in persons under 20. Dr. Simons was certainly prescient in his expressed concern over this disease. How foresightedly he wrote in the concluding paragraph of his book: "It is hoped that this work will so focus attention on its subject that intensified investigation of the etiological aspects of the disease will light the way to prophylactic prevention, that more correct diagnoses will be made earlier in the course of the malady, which will ensure greater possibilities of an increased number of surgically cured cases, and that surgical progress in the treatment of pulmonary neoplasms will also be responsible for reducing the mortality from the disease."

One of the facets of Dr. Simons' professional life, known to only a few of his close associates, was his interest and skill in medical writing. I seem to recall that he worked as a proofreader on different newspapers during his high school and early college days. He continued this type of activity all during his years of practice, when he served as a medical editor for the Yearbook Publishers, Inc., on whose staff his brother, Hi Simons, served as president. To read Dr. Simons' papers in the medical literature is to be struck at once by their clarity, conciseness, and coherence. He would tolerate no word wastage. He knew that physicians inexperienced in writing and unfamiliar with grammar and rhetoric had a natural tendency to be verbose. His blue pencil often cut a lengthy article in half and yet increased its readability and value 100 per cent.

Of course, there was a double motive in Dr. Simons' medical editing. It gave him a chance to improve his own diction and vocabulary while he was enhancing the quality of the publication. But, most important of all, he provided himself with a continuous opportunity to review the medical literature from A to Z. With his remarkable memory and his unquenchable thirst for knowledge, it is no wonder that his medical mind became akin to a 1,000 page book on differential diagnosis.

In 1935 and 1936, Dr. Simons took time off from his practice to join the staff of the Minnesota State Tuberculosis Sanatorium at Ah-gwah-ching, where he sharpened his knowledge on chest diseases; his brother, Dr. John Simons, now of Billings, Montana, carried on at Swanville. In 1942, he moved to Minneapolis and served as chief of Medical Services of the State Division of Social Welfare. Dr. Simons, who succeeded me in this position when I moved to Washington, D.C., brought to this post his great experience in preventive medicine. One of his major achievements was to strengthen the relationships between public health physicians and private practitioners throughout the state.

From 1947 to 1950, he continued in private practice, serving as member of the Board of Directors of Minnesota Medical Services, Inc., (Blue Shield) and, of course, in various capacities in the organizations already mentioned. At that time, 1950, he left Swanville and moved to Edina, Minnesota, where he lived until his death in December 1958.

In 1950, Dr. Simons was persuaded to take the administrative post of executive director of the Minnesota Medical Services, Inc., the Blue Shield plan for the state. The plan was growing and presented many problems that challenged all of his administrative and medical abilities. His was a difficult task, because the program was relatively new and there are chronic differences of opinion among doctors about the cost of medical care and methods of payment. Dr. Simons' positions in the state medical society as a past-president and councilor were invaluable to him in extending and improving the Blue Shield Health Insurance Plan. He continued to insist on a high quality of medical service and used all his talents to achieve that goal. He served the Blue Shield organization faithfully for the remainder of his life.

Dr. and Mrs. Simons have two children, Jack Scott Simons of Minneapolis, born in 1937, and Galen Claire Simons of Minneapolis, born in 1939.

During World War I, Dr. Simons served in the United States Navy as a command ensign on the U.S.S. Kansas, the U.S.S. Arizona, and the U.S.S. Crowninshield and also at the United States Naval Academy. He was an examiner for the United States Veteran's Facility, on the editorial board of *THE JOURNAL-LANCET*, editorial consultant and later coadministrator of Medical Year Books, Inc., Chicago, and coordinator for the National Foundation for Infantile Paralysis in Minnesota during the 1945 epidemic.

As a general practitioner from a small village, he

was particularly proud, and rightfully so, of attaining the high office of president of the Minnesota State Medical Association, which he served diligently and well for so many years. He was a fierce exponent of the private practice of medicine and the rights of the "solo" practitioner. In spite of his accustomed shy and retiring manner, he unhesitatingly waged verbal battle with anyone who dared to suggest that some form of socialized medicine might be a good thing for the country. His arguments were devastating in their logic.

Dr. Simons was a member of the American Medical Association, Upper Mississippi Medical Society, the Morrison County Medical Society, the Hennepin County Medical Society, the Minnesota Trudeau Society, the National Tuberculosis Association, and he was a trustee of the University of Minnesota Medical Foundation. He was Fellow of the American College of Physicians and the American College of Chest Physicians. He served as a member of the Rural Medical School Selection Committee of the Minnesota Medical Association, on the State Certification Body for Public Health Nurses, and as a consultant to the Minnesota State Sanatorium. Dr. Simons served on the staffs of St. Gabriel's Hospital, Little Falls, the Long Prairie Hospital, and the Lymanhurst Health Center in Minneapolis.

Dr. Simons was a unique individual in private life and in his professional life as a general practitioner. He brought honor to the medical profession as a dedicated servant of humanity. He made notable contributions to medicine through his carefully documented observations on general practice and his teaching. I deeply appreciate the opportunity to pay tribute to a true friend, a great physician, and a medical statesman.

AN APPRECIATION

by

C. A. MCKINLAY, M.D.

President of Blue Shield

It is a privilege to have known Dr. Simons. Since his graduation in medicine from the University of Minnesota, he has been an outstanding physician who not only carried on an active practice, chiefly rural, but who found time in the succeeding years to make important contributions to medical literature and to professional organizations. His book on *Primary Carcinoma of the Lung* was the most complete at that time. Dr. Simons found time to carry on clinical research with Dr. Hilleboe in regard to tuberculin testing and case finding in his own community while practicing medicine.

For these and other attainments, his alma mater awarded Dr. Simons, through the regents of the University of Minnesota, the Outstanding Achievement Award in 1956. His accomplishments arose in part from almost boundless energy that led to long hours of labor. If his capacity for work was great, his clearly functioning, analytic mind did not lead

him astray or into bypasses. There was little waste motion. Dr. Simons epitomized the individual practicing physician at his best—one who not only kept abreast of his time but helped to lead his profession. The economics of practice had, in his opinion, a great deal to do with its effectiveness. Basically, this was individual enterprise, which, in medical practice, means free choice of physician. This was medicine without a third party, lay or government interposing itself in the patient-physician relationship. Dr. Simons felt that the physician knew more about the practice of medicine than anyone else, but he was open-minded in regard to public and professional interrelationships. Furthermore, he generously contributed his time to organized medicine, including service as councilor and president of the Minnesota State Medical Association. Administrative capacity was a part of his native gifts. It might be stated that the physical incapacity he was called upon to bear never lessened his drive and accomplishments. Mrs. Simons and their two children were of great assistance to him.

In the light of this background, in 1950, the Board of Minnesota Blue Shield unanimously appointed Dr. Simons as its first medical executive director. During the following years the reasons for his effectiveness became apparent. His qualities of mind, including clear and analytic thinking, often brought out the essential point or heart of the matter, cleared the ground, and helped solve many problems. His maturity and integrity of character, his experiences as a practicing physician, his concept of the social obligations of the profession, including and beyond the question of professional ethics, made him an ideal executive director. He was grounded in the idea of service for the underprivileged and indigent. At the same time, the physician was always considered to be worthy of his hire. Dr. Simons believed that what was good for the physician was, in the long run, good for the public and that their interests are parallel and not opposed.

The patient-physician relationship was always kept inviolate. In his opinion, Blue Shield was formed to support in every way this relationship and was a mechanism which helps physician and patient by the simple expedient of prepayment to maintain private practice. Dr. Simons emphasized the service angle of Blue Shield and of moderation in fee for the low-income group and that human suffering should be alleviated wherever possible, particularly in childhood and old age.

Dr. Simons stands in the memory of those who were privileged to work with him as a friend of mankind and as a statesman in medicine, willing to accept adjustments of mechanism but never compromising with principle. The principle of free choice of physician, of private practice, of noninterference by third parties, whether lay or governmental, were part of his philosophy of life that made him the great physician, humanitarian, administrator, and seeker of the truth that he was.

AN APPRECIATION

by

W. W. WILL, M.D.

Bertha, Minnesota

It has been my very good fortune to have known Dr. Simons for more than a quarter of a century. I have known him very intimately as a friend and as a medical consultant. I can truthfully say that I have never known a medical man who was more fair-minded—fair to the patient and fair to the doctor with whom he was consulting.

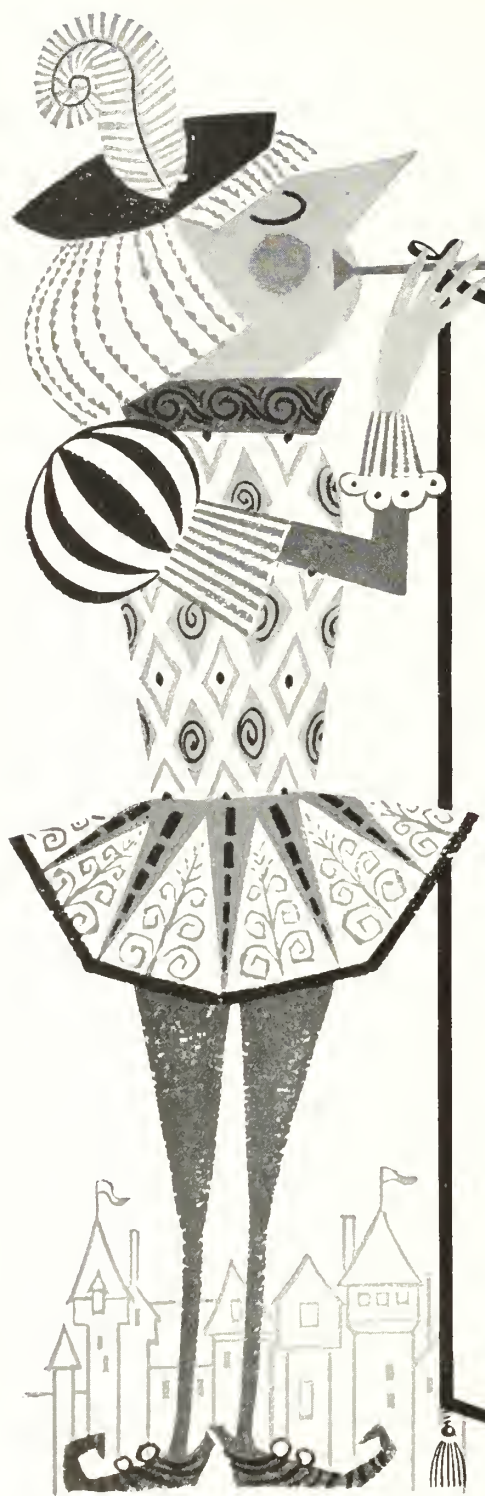
Ed was one of the most studious and hardworking individuals I have ever known. He loved the study and the practice of medicine. He was a firm believer in organized medicine. Without a doubt, his greatest contribution to organized medicine was rendered during the years he was executive director of Blue Shield. A fellow worker of Ed's is said to have remarked, "Ed Simons *was* Blue Shield." We all know that Ed would never have sanctioned such a statement. He always gave all credit to the Board and spoke highly of the cooperation he received.

It is our belief that the majority of medical men of Minnesota are of the opinion that the Blue Shield Board of Directors has done a wonderful job. In eleven years, they have invested \$4,800,000 in United States and municipal bonds to guarantee the solvency and to honor the contracts issued by Blue Shield.

It is too late to tell Dr. Simons of the magnificent work he has done, but, as medical men of Minnesota, let us cooperate with the Blue Shield board and thereby carry out the wish of one who has done so much for organized medicine in our state.

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Book Reviews

The Emergency Syndromes in Pediatric Practice

ALFRED J. VIGNEC, M.D., 1959. New York: *Laudsberger Medical Books, Inc.* 371 pages. \$9.00.

In the preface of this very useful book, the author states that he would limit his discussion to diseases that will brook no delay in treatment. He has quite adequately covered the common and uncommon emergencies that may arise in the pediatric age group. In addition, he has included some conditions, such as congenital cardiac, that usually require time for some degree of diagnostic work-up.

Each section representing related emergencies, such as "Respiratory Tract Emergencies," "Metabolic Disorders," "Poisons," and so on, is well outlined and prefaced by a short discussion of the problems peculiar to each group.

Diagnostic procedures are discussed in detail, and treatment is described. Where therapy is of a medical nature, the author gives the details of the immediate treatment and outlines the long-term management. If surgery is required, the description of the operative treatment is necessarily brief.

Included are sections on fluid and electrolyte therapy, the care of prematures, and the management of certain allergic problems.

The book is a hardbacked, well-bound volume of a size that is easily carried. The illustrations consist of several roentgenograms which, although not always of good clarity, demonstrate the pathology adequately.

Since the book assumes a previous knowledge of the pathology and physiology of the conditions described, it would not be of primary usefulness to the medical students. The intern and resident, especially those interested in pediatrics, would find it helpful, and the pediatrician might find the book handy for reference as a convenient review of the emergencies he may encounter. It would seem that this volume could be most useful to the general practitioner as a reference for diagnosis and treatment. A copy of this work might well be kept in the emergency room of any hospital that has the opportunity to treat pediatric emergencies.

WILLIAM L. BAKER, M.D.
Chicago

Mental Retardation

HANS MANTNER, M.D., 1959. New York: *Pergamon Press.* 280 pages. \$5.50.

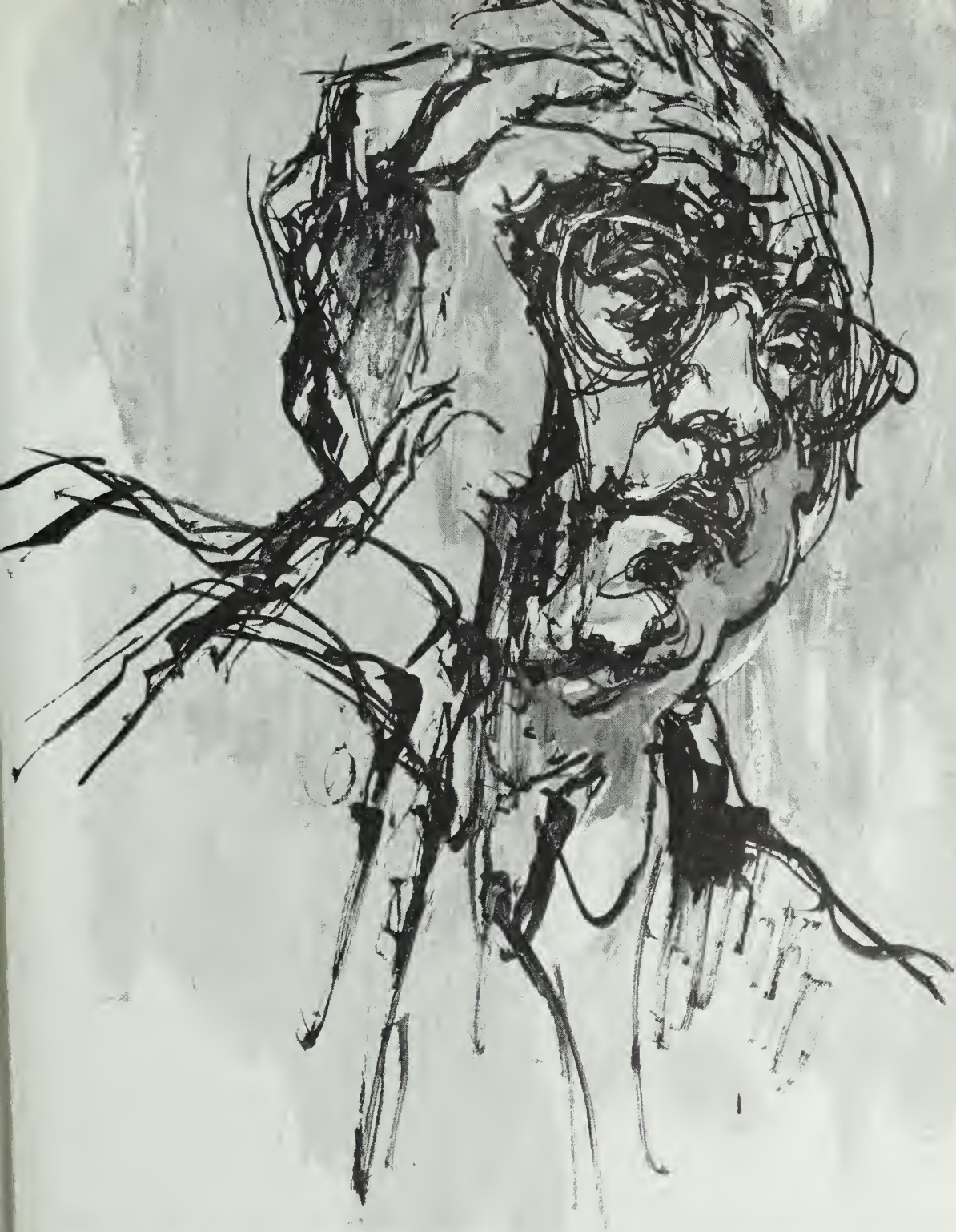
Primarily physiologic in emphasis, this book presents a brief discussion of the architecture, chemistry, and electrical processes of the brain, proceeding thence to brief chapters on diseases, injuries, and hereditary factors which are associated with mental retardation. Mental retardation, the author says, can influence or be influenced by the condition of any organ in the body. Hence, one cannot overlook the importance of metabolism, nutrition, circulation, endocrine balance, kidney and liver functions, and body chemistry in general. Of particular interest to Dr. Mantner is evidence that the action of the whole brain is affected by chemical transmitters in the brain synapses. This interest is reflected in his discus-

sion of the physiologic characteristics, symptoms, and treatment of the mentally retarded. He reports the experimental use of drugs which prevent the destruction of the chemical transmitters in the brain, reinstate chemical balance, stimulate the secretion of the necessary chemicals, or act upon the reticular relay system of the medulla, hypothalamus, and thalamus, which apparently regulate brain activity. Modest claims are made for drug therapy in the improvement of some patients of low mentality. The limitations, dangers, and obstacles involved in this type of treatment are clearly defined. Diet seems to offer some hope for improving the intellectual functioning of the very young child with certain kinds of imbalances, for example, a high phenylalanine level in the blood. However, the results of research in this area are somewhat contradictory. Iodized salt for Cretinism; early blood transfusions for erythroblastosis; and surgery for epilepsy, hydrocephaly, or focal brain damage are said to be successful in some cases. Prevention is urged as the most effective means of controlling brain damage from accidents, poisoning, infections, contagious diseases, and birth trauma. Great care is recommended for infants of premature births, cesarean section, prolonged labor, or very rapid births. Fever therapy appears useful in a few cases of retardation. Precautions are urged to avoid damage to the unborn child from irradiation. The importance of prophylaxis and early treatment is stressed. Prenatal consultation and sterilization of the unfit are cautiously explored as rather remote possibilities for restricting the number of retarded produced by familial and genetically determined influences.

Education and psychotherapy are briefly mentioned as means of treatment. Psychoanalysis, as treatment, is rejected, although the author asserts that psychoanalytic theory offers insights into the needs and behavior of the mentally handicapped. A relationship of trust and friendship between patient and therapist is deemed essential to progress. Surprisingly, no mention is made of Rogers' client-centered therapy or play therapy as a useful method for the moderately retarded person with emotional problems. This approach has proven quite helpful in some such cases. However, Dr. Mantner does record one case in which improvement might be due to the removal of a child from a cold emotional climate to one in which love and intimate human contact were provided. He ignores the work of Bruno Bettelheim with the pseudo-retarded and the profound contributions of Kurt Goldstein to the treatment of the brain-damaged.

With respect to diagnosis, the author discusses symptoms and characteristics of Mongolism, epilepsy, encephalitis, spasticity, athetosis, oxygen deprivation, oxygen poisoning, embryopathy, microcephalus, and Heller's disease as well as a variety of other deviations, injuries, and diseases. Psychologic tests for measuring intelligence and personality factors are briefly described. Mantner is careful to point out that the same symptoms can come from different causes and that the same source can produce different symptoms, and he warns against making assumptions from insufficient evidence. He observes that brain damage does not necessarily result in mental retardation but makes no reference to perseveration nor to the catastrophic reaction frequently manifested by brain-damaged patients.

(Continued on page 32A)



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C I B A
SUMMIT, N. J.

BOOK REVIEWS

(Continued from page 30A)

This book is actually a collection of lectures given by the author to the staff of the Pineland Hospital and Training Center in Pownal, Maine. Each of the 25 short chapters is abundantly documented. Some of them, abbreviated and condensed as abstracts, are followed by as many as 70 or 80 references. In spite of these mildly confusing facets and a rather bewildering array of specialized professional terms, the book is quite readable. The accounts of research on brain chemistry and drug therapy are especially interesting. Probably this volume would be most useful as a resource reference for students of medicine or psychology and for people already engaged in work with the mentally retarded.

JEANNE A. WOOLF

Counseling Psychologist

The Counseling Clinic, Honolulu

Automatic Ventilation of the Lungs

WILLIAM W. MUSHIN, M.A., L. RENDELL-BAKER, M.B., and PETER W. THOMPSON, M.B., 1959. Springfield, Ill.: Charles C Thomas. 349 pages.

This book is written by anesthesiologists and for anesthesiologists, although anyone having responsibility for the care of patients requiring artificial or assisted ventilation of the lungs will find it interesting and helpful. The book divides itself naturally into 2 sections. The first deals with the physiologic and clinical aspects of controlled breathing and the principles utilized in auto-

matic, mechanical respirators. The second and much larger section of the book describes in some detail practically all of the mechanical devices now on the market for artificial ventilation of the lungs. The various machines are divided into four groups: time-cycled ventilators, pressure-cycled ventilators, volume-cycled ventilators, and ventilators employing some combination of these principles.

The first 92 pages of the book deal with physiologic and clinical aspects of artificial ventilation of the lungs and with the basic principles underlying mechanical devices used for this purpose. The roughly 250 remaining pages constitute a handbook on automatic mechanical respirators. It is this latter portion of the book that most clinicians will find valuable. Each description is well illustrated with diagrams showing the operation of the instrument and, in most cases, with actual photographs of the device. A functional analysis for each machine is included with the description.

The introductory section of the book emphasizes the many situations in clinical medicine in which assisted respiration is necessary. It also rightly emphasizes the fact that during anesthesia, contrary to the situation that exists with air breathing, when a high concentration of oxygen is present in the inhaled mixture, carbon dioxide may accumulate to toxic levels from inadequate pulmonary ventilation in the face of normal or high oxygen tensions. These are areas in which the trained anesthesiologist and pulmonary physiologist are well oriented, but a review and reemphasis of this material is not amiss.

E. B. BROWN, JR., M.D.

St. Louis

(Continued on page 34A)

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BOOK REVIEWS

(Continued from page 32A)

The Physiology and Treatment of Peptic Ulcer

J. GARROTT ALLEN, *Editor*, 1959. Chicago: University of Chicago Press. 236 pages. Illustrated. \$7.50.

This small book is a collection of monographs by former associates and students of Dr. Lester R. Dragstedt. Each paper covers in minute detail its subject, whether it be research, medicine, or surgery. Thus, for the clinician, we find that it is quite easy to get lost in the details of the research that has been done on peptic ulcer. Knowing such might help in treating an ulcer, but I doubt it.

The final 2 pages of the book contain a short article by Dr. Dragstedt entitled "What Would I Do if I Had an Ulcer?" This article is better than all the rest of the book together.

W. RALPH DEATON, JR., M.D.
Greensboro, North Carolina

Gouty Arthritis and Gout

THOMAS E. WEISS, M.D., and ALBERT SEGALOFF, M.D., 1959. Springfield, Ill.: Charles C. Thomas. 182 pages. Illustrated. \$7.50

This book tells the story of gout very well. Not only the very fine introduction but the whole book is flavored with the medical history that is so interesting to most students. The section on physiology is very detailed and well written and should be stimulating to even the graduate student. The section on pathology is extremely well illustrated. The photographs are of the highest quality and numerous. The clinical aspects of the disease are apparently backed up by a more than adequate clinical experience. The section on treatment is most complete and up to date, including zoxazolamine in the armamentarium.

One of the outstanding features of the book is the list of almost 400 references on gout. The index is most adequate. I think this book should be in every hospital library where there is a teaching program. It should also interest the general practitioner and the internist. It is a must for anyone having a significant number of arthritic patients in his practice.

LESTER E. WOLD, M.D.
Fargo, North Dakota

A Textbook of Surgical Physiology

R. AINSLIE JAMESON, M.B., and ANDREW W. KAY, M.D., 1959. Baltimore: Williams & Wilkins Co. 610 pages. Illustrated. \$11.00.

This new textbook has been prepared by the authors with the postgraduate medical student in mind and in the belief that there is need for a modern text dealing with surgical physiology. The authors were lecturers in surgery at Glasgow University when they developed the idea of writing. Each author is well recognized in the field of surgery, and each has contributed in the fields of surgical physiology and pharmacology. The text is well prepared for postgraduate students in surgery and should aid review for basic science and board examinations. Its scope and organization have qualified it as a valuable reference source for undergraduate students. The 26 chapters well cover the field of surgical physiology. The chapters on fluid balance and metabolic responses to trauma well summarize these subjects, and the chapter

on biologic effects of radiation is interesting. All chapters closely associate the basic principles of physiology with the practical problems of their application to surgery. This is a useful and important contribution to the surgical literature.

KEITH S. GRIMSON, M.D.
Durham, North Carolina

Clinical Dermatology for Students and Practitioners

HARRY M. ROBINSON, JR., M.D., and RAYMOND C. V. ROBINSON, M.D., 1959. Baltimore: Williams & Wilkins Co. 242 pages. Illustrated. \$8.50.

The authors of this book have done an excellent job in condensing the practical and important aspects of clinical dermatology into a compact volume written primarily for medical students and general practitioners. Part 1 of the book is devoted to short discussions of anatomy, physiology, etiology, diagnostic procedures, mycology, allergy, venereal diseases, and therapy. Occupational dermatoses and psychosomatic aspects of dermatology are briefly mentioned. Part 2 deals with morphologic dermatology. Specific diseases are classified according to primary lesions and are briefly discussed. Chapters on diseases of the skin appendages and tropical and peripheral vascular diseases are also included. The book is well illustrated with excellent black and white photographs and diagrams; however, there is no bibliography. In general, this volume is well organized and should be useful as a text for students and a quick reference for interns, residents, and practitioners.

ELMER HILL, M.D.
Minneapolis

Pediatric Neurology

STANLEY S. LAMM, M.D., 1959. New York: Landsberger Medical Books, Inc. 482 pages. Illustrated. \$12.90.

This volume represents the distillate of thirty-five years of experience by a clinical pediatrician in the field of neurologic disorders of childhood. Although to some extent the presentation follows traditional textbook form, the volume seems intended for, and certainly has its greatest usefulness in the hands of, a busy practitioner well qualified in pediatrics. Considering the rapid growth and great complexity of the field of pediatric neurology, the author has compressed into a relatively small volume a great deal of useful information. He has avoided exhaustive discussions of controversial points and presented his facts clearly and succinctly. A practical bibliography follows the presentation of each category of neurologic disorder. The references for the most part are useful in that they are available in any adequate medical library.

The author has clearly been plagued by the problem of attempting a logical arrangement within a diffuse and difficult field. His initial chapter on genetics suffers from lack of direct neurologic reference, although this appears later in the discussion of specific disease entities. It is to be hoped that a second edition might be improved by some categoric rearrangement and a more comprehensive index. The illustrations are adequate, but the book would not suffer if they were omitted.

This is a useful addition to the bookshelf of the busy practitioner of pediatrics or the generalist interested in childhood disorders.

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Providence, Rhode Island

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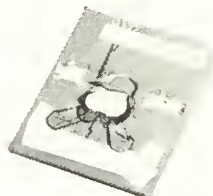
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News Briefs . . .

North Dakota

DR. H. D. BENWELL has succeeded Dr. R. E. Mahowald as chief of staff and president of the advisory board of St. Michael's Hospital in Grand Forks. A graduate of the University of Manitoba, Dr. Benwell has been a member of St. Michael's staff since 1923, at which time he became associated with Drs. H. M. Wheeler, R. D. Campbell, and G. M. Williamson.

DR. ERWIN LENGYEL, a native of Austria and a naturalized American citizen, has been named the third certified psychiatrist to the staff of the Jamestown State Hospital. A graduate of the University of Vienna, Dr. Lengyel served a five-year residency in medicine and surgery at Franz Joseph Hospital in Vienna and was medical director of the American Presbyterian Hospital in China from 1939 to 1949. Since his arrival in the United States in 1952, he has been a member of the staff of the Utica, New York, State Hospital.

DR. E. R. WASMILLER of Wahpeton has been elected president of the medical staff of St. Francis Hospital for the coming year. Other new officers are Dr. P. H. Engstrom of Wahpeton, vice-president, and Dr. J. H. Singbeil of Breckenridge, secretary.

DR. R. D. McBASE has been named president of the Devils Lake District Medical Society, to be assisted by Dr. Stuart Cook of Rolette as vice-president and Dr. L. F. Pine of Devils Lake as Secretary. New delegates to the State Medical Association Convention are Drs. D. W. Palmer of Cando and J. H. Mahoney of Devils Lake.

DR. C. W. SCHOREGGE, DR. F. F. GRIEBENOW, and MR. SPENCER S. BOISE were honored by the Quain and Ramstad Clinic in January for service totalling 122 years. Dr. Schoregge, a native of Sleepy Eye, Minnesota, came to Bismarck forty-four years ago after graduating from the University of Michigan, interning at Asbury Hospital in Minneapolis, and practicing medicine in Henderson, Minnesota. He was chairman of the Quain and Ramstad Clinic until 1955 and is a member of the American College of Surgeons and the Sixth District Medical Association as well as being a diplomate and Fellow of the American Board of Surgery and a Fellow of the International College of Surgery. Dr. Gribenow, who received a fifty-year service pin at the last annual convention of the North Dakota Medical Association, was graduated from the University of Minnesota College of Medicine and Surgery in 1909. After a year of service at the Bismarck Hospital, he joined the Quain and Ramstad Clinic, his principal interest lying in the field of dermatology. He is a member of the American and North Dakota medical associations and the Sixth District Medical Society. Since his retirement, he has done occasional work in general practice and public health. Spencer S. Boise became business manager of the Quain and Ramstad Clinic in 1922 after graduation from Fargo College. Although he is now semi-retired, he intends to spend some time at this position as long as possible. He is a member of the National Clinic Managers and the Bismarck Association of Commerce and has served as president of both.

(Continued on page 39A)

The Journal Lancet

SERVING THE MEDICAL PROFESSION OF MINNESOTA,
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The First Ninety Years

J. ARTHUR MYERS, M.D.

Minneapolis

CHRISTOPHER CARLI of Heidelberg, Germany, who opened an office at the present site of Stillwater in 1841, was the first civilian general practitioner to settle in the area that became Minnesota. He had been preceded only by missionary and military physicians.

With the passage of a bill creating the territory of Dakota in 1861, Dr. William Jayne, a practicing physician from Springfield, Illinois, was appointed governor of the new territory. The next year, Dr. W. D. Dibbs was the first officially appointed army doctor in that part of the territory which became North Dakota.

Because the physicians who migrated to what became North and South Dakota and Minnesota were dependent upon only a few national medical publications, they felt the need of an area medical journal. Therefore, on February 1, 1870, Dr. Alexander J. Stone of St. Paul proposed that a medical journal be published, and the members of the state medical association promised to subscribe.

The first issue appeared in June 1870 under the title of *Northwestern Medical and Surgical Journal*. After two years, publication was suspended because of lack of financial support. In October 1881, the journal appeared under the name of *Northwestern Lancet*; the name was changed to THE JOURNAL-LANCET on January 1, 1912.

No school of medicine, nursing, dentistry, or veterinary medicine existed in what was known as the Northwest when THE JOURNAL-LANCET was established. No law regulated the practice of medicine, and there was no State Board of

Health. In due time, however, hospitals, professional schools, and so forth began to appear, thereby increasing the need for a medical journal.

When THE JOURNAL-LANCET was founded, a great number of persons still believed in spontaneous generation, as they had not accepted the work of Pasteur, Villemin, and Lister. This medical magazine was in the field as the scene unfolded. Beginning with anthrax, one pathogenic microorganism after another was isolated. Causal relationships between certain microorganisms and diseases were well documented. That period has been referred to as one of the glories of nineteenth century medicine.

Those persons in charge of THE JOURNAL-LANCET saw antitoxins for diphtheria and tetanus come into existence and, later, immunizing agents for diphtheria and typhoid fever. They saw intestinal disorders, such as cholera infantum, conquered as a leading cause of death. They saw the discovery of the fact that an intermediary host transmitted a protozoan from animal to animal, causing tick fever in cattle; this opened the door for the discovery of the cause of malaria and yellow fever. They were present when the first attempts were made to treat many conditions surgically and recorded the ever-improving methods in anesthesia. They saw the discovery of vitamins and the unfolding of this field, as well as that of allergic conditions. They saw the appearance of the x-ray and the bronchroscope. They saw veterinarians attack diseases of animals transmissible to man.

Although specific treatment for most bacterial

diseases was not developed for several decades, THE JOURNAL-LANCET was still reaching offices of many physicians regularly and, in due time, was carrying news about such drugs as sulfonamides, sulfones, para-aminosalicylic acid, isoniazid, and antibiotics, as well as early information about surgery of the chest wall and, later, pulmonary resection and open heart operations. Possibly, the period beginning about 1935 may always be regarded as the glory of twentieth century medicine.

Articles in this Ninetieth Anniversary issue were prepared by experts in the subjects under discussion, showing the tremendous progress that has occurred and bringing to date the current state of knowledge and development, along with a look into the future.

The subject of genetics in medicine, the importance of which has become so widely recognized, is discussed by Dr. Edwin J. Grace under the title of "Microbial Evolution and Antibiotic Therapy." Dr. Grace presents facts that are alarming with reference to previous indiscriminate use of antibiotics. If any reader of THE JOURNAL-LANCET is prescribing and administering penicillin or other antibiotics for conditions for which there is not well-documented evidence of efficacy, the reading of this paper should halt him until he has learned "how to use them with safety and unalloyed success." Publication of this paper enables readers everywhere to know of the harm that may already have been done and the importance of the medical profession's instituting an immediate campaign among producers of antibiotics and physicians to stop their use except when specifically indicated.

One function of THE JOURNAL-LANCET has been to keep facts before its readers in order to prevent complacency during and following outstanding accomplishments. For example, for a time, diphtheria was at such low ebb that most physicians, while examining persons with symptoms and findings which in the past would have immediately aroused suspicion, forgot to look for the specific organism. This oversight resulted in a considerable number of deaths because, by the time the diagnosis was made, the individual had died or was so near death that antitoxin was unavailing. Despite all that has been learned, we have not eradicated a single communicable disease. For example, in this country alone, more than a thousand cases of diphtheria are reported and several hundred deaths occur annually. Although methods of eradication of smallpox have been known since 1796, 400,000 cases are still reported in the world each year. Despite

the fact that more incapacity and deaths are now caused by tuberculosis than by all other communicable diseases combined, in this area in the United States and in the world as a whole, complacency among physicians has become alarming. Thus, a series of articles on communicable diseases has been appearing in THE JOURNAL-LANCET for nearly two years, to keep these conditions in the minds of physicians and to bring to them last-minute information.

So much knowledge has been added to the physician's armamentarium over the past few decades that it has become physically impossible for anyone to read the huge number of articles on even a single phase of medicine appearing in the 12,000 medical journals of the world. Therefore, THE JOURNAL-LANCET provides a series of brief articles, each of which brings the reader up to date in a few minutes, on the numerous diseases and conditions with which our readers have to cope.

Throughout the ninety years of its existence, numerous efforts have been made through the pages of THE JOURNAL-LANCET to insure close cooperation between various professional groups and full support of local state and national boards of health, the nursing profession, the veterinary profession, and so forth. The great importance of the veterinary profession in controlling and eradicating animal diseases transmissible to man has often been emphasized.

Length of life has been prolonged by controlling diseases and conditions that formerly were destructive among the young, particularly communicable diseases and infections of the gastrointestinal tract. In 1870, the average length of life in this country was approximately forty years. The life expectancy for children born from 1900 to 1910 was fifty-one years for girls and forty-eight years for boys, whereas girls and boys born in 1960 have a life expectancy of seventy-three and sixty-eight years, respectively. In Minnesota, the age group from birth to 19 years, which constitutes about 36 per cent of the population of the state, accounted for 45 per cent of the population increase during the past decade. Among persons of 20 to 64 years of age, who constitute about 55 per cent of the population, a 43 per cent increase was reported. Persons of 65 or over, constituting 9 per cent of the population, accounted for 11 per cent of the total population increase.

Less has been accomplished in controlling diseases and conditions prevalent among elderly persons. However, some progress has been made even in this area.

Accidents took about 91,000 lives in this country in 1959. In this issue of THE JOURNAL-LANCET, Dr. Stewart Thomson has a splendid presentation on some aspects of the problem of farm accidents in which he calls attention to the role of physicians.

Brief biographic sketches of physicians who have made outstanding contributions in practice teaching, research, administration, education, and so forth have been published, beginning with H. Longstreet Taylor of St. Paul in January 1932. Glimpses into the lives of these physicians have proved so inspiring that such profiles have appeared regularly for some time.

Over the past ninety years THE JOURNAL-LANCET has published thousands of articles by

physicians who wished to present results of their research, clinical observations, and so forth to their fellow practitioners. New discoveries, inventions, and methods have been announced in order that physicians might become more helpful to those whom they serve, individually and collectively. With equal fervor, THE JOURNAL-LANCET has attacked imposters, charlatans, quacks, and nostrums and has supported every worthy project designed for the betterment of health.

The future goal is not only to hold and increase the gains already made but also to support methods aimed at control of degenerative diseases, thus increasing the comfort, happiness, and efficiency of people everywhere.

PEPTIC ULCER in children shows signs and symptoms varying with the age of the patient. Sudden hematemesis or melena is usually the first manifestation in children under 2 years of age. In the neonatal period, the ulcer either heals quickly or causes rapid death.

In 2- to 9-year-old children, periumbilic or epigastric pain is associated with nausea and vomiting. Anorexia, pain before breakfast, and temper tantrums are also present. Symptoms are more typical and chronic in older children. The rhythmic hunger pain is usually seen only after age 15. Ingestion of food and alkali does not appear to give as much relief to children as to adults, but pain may subside after vomiting. Heartburn is a rare symptom in children.

Because large ulcer niches are seldom demonstrable, radiologic diagnosis must often be based upon pylorospasm and duodenal deformity.

Although hyperacidity is not common in children, the blood pepsin level may be elevated. Childhood ulcers are thought to be caused by environmental conflict or insecurity combined with an inherited predisposition. Improvement of the environmental situation and increased parental understanding aid recovery.

Rigid diets have unfavorable psychologic effects on most children. Instead, patients are advised to eat frequently, drink milk between meals, and avoid only the obviously harmful foods.

When perforation or hemorrhage makes surgery necessary, hemigastrectomy and vagotomy appear to be preferable. Every effort should be made to avoid surgery in patients under 16, since subtotal gastrectomy may seriously retard development. If gastroenterostomy is done, vagotomy should probably always be included in order to prevent marginal ulcers.

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Microbial Evolution and Antibiotic Therapy

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IN THE NINETY YEARS since the founding of THE JOURNAL-LANCET, history has recorded events which were beyond the realm of credulity of those men of the mid-nineteenth century. At times in the last decade or two, even we "moderns" have been hard put to believe the things we see and hear and read about. However, it is well to reflect on these words of Arnold Toynbee: "What will be singled out as the salient event of our time by future historians, centuries hence, looking back on the first half of the twentieth century . . . Not, I fancy, any of those . . . events which occupy the headlines of our newspapers and the foregrounds of our minds . . . the slower, impalpable, imponderable movements that work below the surface and penetrate the depths . . . in the end, make history, and it is they that stand out huge in retrospect, when the sensational passing events have dwindled, in perspective, to their true proportions."

From our vantage point today, it might seem that penicillin is one of medicine's greatest contributions to the twentieth century, but if we look below the surface of this "great" event, we find a "deeper, slower" movement. We are speaking, of course, of the emergence of resistant bacterial strains following the use of modern antibiotics.

This emergence and perpetuation of resistant bacterial pathogens is among the gravest problems confronting the world today and is a direct threat to future generations. The problem is man made, and all present efforts to control the mounting mortality and morbidity rates from these pathogens are proving inadequate. However, some progress might be made if we clearly understood the biologic phenomena involved in this ominous epidemic of bacterial mutants which threaten not only man's welfare but his very existence.

While we were curing the incurable and curbing the spread of the world's greatest killer, tuberculosis, we disregarded genetics and spread

lethal mutants in hospitals and homes. The microbial geneticists have time and again pointed out to the medical profession the dangers of mutation developing during antibiotic therapy, and they have laid down therapeutic methods to prevent and control this mutation.

Vernon Bryson, a geneticist, has stated that "multiple chemotherapy is at present the most efficient method for preventing the establishment of resistant strains, but it has certain potential limitations, many of which also apply to the use of drugs singly. These limitations result from the growth of resistant clones before the onset of antibiotic therapy. . . . Through no intrinsic fault, multiple chemotherapy must often cope with bacterial populations that have previously been allowed to develop consecutive resistances by the unsuccessful application of several antibiotics in sequence or in ineffective combination."

With the introduction of chemotherapy at the start of the twentieth century, it was observed that both salvarsan and, later, sulfa became inefficient, fundamentally because of the emergence of resistant strains. The use of penicillin alone achieved dramatic results at first, but later the drug's therapeutic effect became more and more limited. The obvious and elementary evidence and the warnings of geneticists concerning the epidemic pattern of bacterial mutation were unfortunately ignored, and the profession must now, in 1960, appraise these new and infinitely more lethal bacterial mutants which emerged almost simultaneously in the mid-twentieth century with the exploitation of the atom.

A notable development in transition in atomic energy research to biogenic concepts for medical therapy by H. Vogel at the Argonne Laboratories is the realistic experimental appraisal of the damage of atomic energy and the dramatic reversal when multiple chemotherapy, streptomycin plus cystine and bone marrow, is used to counteract the effects of lethal doses. What a tragedy it would be if, as we broadened the benevolent effects of chemotherapy on the one hand, we spread malignant concomitants by wrong use of these truly wondrous drugs.

A very superficial study of medical practice

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indicates the magnitude of the lag in integrating genetics with clinical practice. The problem has reached such proportions that the surgeon and physician and any moderately well-informed layman can foresee the lethal consequences of the microbial mutant epidemic. In the evolutionary world, the staphylococcal mutant is a microbial monster which is man made. This medical and evolutionary phenomenon is adequate testimony to our inability or unwillingness to grasp genetic fundamentals in therapy.

Confronted as we are now with a world-wide biogenetic epidemic, for which we are unwittingly but nevertheless morally responsible, it is obvious that many of the older therapeutic concepts that antedated the discovery of penicillin must be adapted by man to the resurging world of microbial evolution. The physician responsible for man's welfare, and possibly for his survival, must understand why this bacterial mutant emerged, and he must have an understanding of this species of the staphylococcus organism in relation to the life of man. If man is to survive on this planet, we are compelled to view the genetic phenomenon of man's evolution.

A review of the current literature on bacterial mutation and resistance by most investigators shows a regrettable tendency to confine the problem to the last decade instead of seeing this recent microbial enemy of man as still another epoch in his centuries-old struggle for survival.

Speaking before a group of the world's most distinguished geneticists, Mayr asked, "Where are we?" and then proceeded to appraise the stage of evolution in the 1959 time-space world. As clinicians, we should ask ourselves, "Where are we?" Since a fundamental of good medical practice is always a review of errors, we in American medicine today should eschew any feelings of chauvinistic superiority in order to examine the gains and losses of medical technology in the history of the last two decades.

We in the medical profession must stop our forward rush into the bigger and brighter future and take time to integrate the discoveries and theories of the past with those of the present. Thus, to cope with the bacterial mutation problem, we must turn to Darwinian concepts of evolution and Mendelian genetic principles. The study of genetics must be a basic part of our medical curriculum, and research in evolutionary phenomena must be carried out. Furthermore, as a first step, we must recognize that the phenomenon of bacterial resistance or bacterial mutation to antibiotics can be controlled with

satisfactory clinical results if we use combinations of antibiotic therapy.

The rediscovery of Mendelian genetic principles in the first decade of this century has been almost completely ignored by the physician, whereas the agronomist has used this knowledge to grow hybrid corn, breed cattle, and achieve agricultural production which is admired throughout the world. Whereas the physician continues to contribute to the spread of a world-wide epidemic of resistant bacterial strains, the agronomist has been pursuing the agricultural application of genetics for nearly half a century, so that agrarian development today is one of our chief resources.

One may inquire why, with the problem so menacing, we remain indifferent. The answer lies in the sophisticated, complacent atmosphere that has existed in American medicine for the last three decades. It was well stated by the great German physicist, Max Planck, who said: "A new scientific truth does not triumph by convincing its opponents but because its opponents die and a new generation grows up that is familiar with it."

Since American medicine has been so intimately related to the technologic development of our Western civilization, we have had an unfortunate tendency to feel that at some appropriate period when a problem in medicine becomes acute, if we can only get enough public support and the aid of governmental finance and all health agencies, any health problem can be solved—the so-called crash program. This approach will not lead to the solution of our present problem, and meantime the epidemic will spread and new mutants will develop. As physicians and investigators, we must apply the basic genetic concepts in therapy that we have to date flagrantly violated in spite of the repeated warnings of our Western (Mendelian) geneticists.

The physician must be a catalyst to integrate biogenic concepts with therapeutic technics in present and future medical problems, and he must also pinpoint the source of that which could become our greatest medical problem—the lethal bacterial mutant. As we move from a primary national interest in atomic medicine to biogenic medicine, the physician of the future can see new opportunities and perceive his grave historical responsibilities if he reads *The Guidance of Human Evolution* by H. J. Muller, the Nobel Prize winner in medicine and physiology for 1946.

Here, with great clarity, is presented a description of man's biogenetic differentiation and emergence from the microbe over a span of bil-

lions of years, and just as clearly presented is the frightening possibility that this primitive struggle of past eons may have to be repeated unless we use for our survival the biogenic tools we now have but which, because of ignorance or complacency, we are not using. Therapeutically, the health and longer life of man are synonymous, first, with an understanding of genetic mutations and, second, with a knowledge of how to control them.

For the medical biologist apprehensive for the future health of man in this fast-shrinking world, there were no events of great clinical significance in the fall of 1959, the year of Darwin's centennial anniversary. However, Stanford University School of Medicine, aware of the need for medicogenetic integration and recognizing the importance of the medical discipline of genetics, has appointed to the staff of the medical school at Palo Alto the Nobel Prize winner in genetics, Joshua Lederberg. We may anticipate the establishment of some new education fundamentals with such opportunities now opening up in medical schools. Likewise, sound clinical contributions based on genetic concepts have accumulated in reports from all over the world since World War II, which may herald the beginning of a new era of genetic medical practice.

In the first conscious encounter with bacterial mutants, we must ask ourselves whether we have lost the first battle—and, in my opinion, we have. Now we must determine whether medicine can retrieve this loss or whether we will continue to retrogress with the expansion and perpetuation of these man-made pathogenic mutants until, at some point in the immense time scale of evolution, we are finally defeated by the microbe. Of course we should not, and with the earlier integration of genetic thinking in clinical medicine and the recent, more formal educational trends personified by Lederberg and Muller, medicine has a foundation it never had before and one which it cannot afford to ignore if it is to fulfill the humanitarian responsibilities associated with medical practice down through the ages.

To awaken the medical profession to this startling situation in which the future of mankind may be threatened, it would be difficult to improve on the following summation by Muller in which the picture is clearly presented:

"Through billions of years of blind mutations, pressing against the shifting walls of their environment, microbes finally emerged as men. We are no longer blind; at least, we are *beginning* to be conscious of what has happened and of what may happen. From now on, evolution is what we make it, provided that

we choose the true and the good. Otherwise, we shall sink back into oblivion. If we hold fast to our ideal, then evolution will become, for the first time, a conscious process. Increasingly conscious, it can proceed at a pace far outdistancing that achieved by trial and error—and in ever greater assurance, animation, and enthusiasm. That will be the highest form of freedom that man, or life, can have."

The nature and extent of bacterial mutation following the use of antibiotics for infectious diseases has been a compelling reason for the physician to expand his knowledge of genetics and evolution in order to advance the welfare of man and foster an environment of health both intrinsically and extrinsically—to establish in man the steady physiologic state or homeostasis of Cannon. To focus our thinking on genetic perspectives, I. M. Lerner states in his book *Genetic Homeostasis* what should be a preamble to every medical publication:

"One of the most important contributions made by Walter B. Cannon to the field of physiology was the development and elaboration of the concept of physiological homeostasis. He has defined homeostasis as the totality of steady states maintained in an organism through the co-ordination of its complex physiological processes. By extension it may be said that homeostasis refers to the *property* of the organism to adjust itself to variable conditions or to the self-regulatory mechanisms of the organism which permit it to stabilize itself in fluctuating inner and outer environments. . . . Undoubtedly, the early notions on the subject tended to view homeostatic mechanisms from a crude vitalistic standpoint. It is to the richly deserved credit of Cannon and his followers that this apparently mysterious and mystical property of biological organisms has been reduced to terms of interactions between constituent parts and functions. Needless to say, final answers to the problems of physiological homeostasis are by no means available. Indeed, the origin of biological feedback mechanisms is in itself a question of central interest in the study of evolution. Their existence is recognized, the largely mechanistic basis of their operation is accepted, but their increasingly complex development from the lower to the higher forms of life still presents the focal problem of evolution of adaptations. That physiological homeostasis is essentially the fundamental adaptation was recognized even before the term was coined. . . ."

Some basis for an amicable adaptation is a prerequisite for man's continued existence biologically and socially. Furthermore, this existence must be not merely life but the life of man evolving to a superior being with the physician as an active agent in this process. To fulfill his role, the physician must utilize all his knowledge and tools, and among these our biogenic therapeutic assets must be recognized as indispensable first to the preservation and, ultimately, to the progress of man in the evolutionary scheme.

One final point—we are all aware that, in the present contest between the United States and the Soviet Union for the loyalties of the world's backward nations, we Americans are using our great medical facilities and technology to assist the peoples of Asia and Africa. These peoples have a belief in the curative effects of antibiotics which borders on the magical. Yet, in these countries, the conditions under which the drugs are used are of necessity primitive. A failure of antibiotic technics, which are definitely identified with Western medicine, particularly American, could have deplorable long-range effects on our relations with these peoples. Thus, we have a far-reaching responsibility not only to use these

drugs correctly ourselves but also to lead the way in ensuring that others who learn our technics are not mistaught or misled through our errors.

SUMMARY

A new lethal microbial parasite has emerged as a mutant in the last decade which we have been unable to control or destroy. We must recognize the fact that man is in constant mortal combat for survival against his oldest enemy, the microbe, and that, while we do have new weapons in the modern antibiotics, we must learn from the microbial geneticist how to use them with safety and unalloyed success.

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DIFFUSATES FROM burned skin circumventing the circulation of rats contain a toxin fatal to mice and rats when administered subcutaneously, intravenously, intraperitoneally, or intracerebrally. Rapidity of death is determined by the total dose and route of administration. The toxin is dialyzable, heat stable, and partially precipitated by 80 per cent ethanol. Its chemical composition is unknown, but it is known to contain peptides, polynucleotides, hexoses, and pentoses. Histamine, bradykinin, adenylyl compounds, and salts are independent of the burn toxin but are thought to contribute to its lethal effect.

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Drug Therapy: 1870

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THE JOURNAL-LANCET is celebrating its ninetieth birthday. Since 1870, this publication has served the medical profession of the Northwest. Everyone in medicine should offer his sincere congratulations to the editors and their assistants and to the publishers for this outstanding record. At a time like this, it is fitting to pause and consider what has happened in the various medical disciplines during this time interval. This should give everyone a feeling of gratitude on the one hand for the progress that has been made. On the other hand, as we look into the future, a sense of responsibility to medicine should develop within us all so that we may help with dedicated and sincere efforts to continue the record of progress.

Perhaps no discipline in medicine can show a more remarkable picture of progress than that of the basic science of pharmacology. Ninety years ago, this science had just been born—and then only in Europe. Only a few schools had recognized this field as an experimental science for the purpose of studying the actions and uses of drugs. In America, the first department of pharmacology was established at the University of Michigan in 1891 under Professor John J. Abel. In 1893, he was called to Johns Hopkins University. At this University, in 1899, he announced the isolation of the first hormone, to which he gave the name of epinephrine as its benzoyl derivative. This was the beginning of a development in therapy, which, even to this date, has not reached its final stages. As a result of the early pharmacologic studies, particularly in Europe, the *materia medica* approach—a purely descriptive approach to drugs—was being questioned, and therapeutic nihilism became more and more dominant. However, once started, the discipline of pharmacology continued to advance slowly and painfully. Within the last twenty-five years, with the help of the synthetic chemist, the rate of discovery and introduction of new drugs has become exciting and, at times, even hectic.

A physician knows full well that the basic knowledge on standard drugs is enclosed in the

Pharmacopoeia and that in America this book is commonly called the *United States Pharmacopoeia*. All physicians know, furthermore, that, together with their colleagues in the state medical associations, they have a significant responsibility for editing this book. At present, delegates from the state medical associations and other institutions gather every five years for the purpose of revision. Formerly, they met every ten years. Preparations are being made today for a revision that will bring out volume XVI of the U.S.P. The first meeting for this purpose was held in Washington in March 1960. Even though a physician does not study this book every day in practice, he is well aware that without it as a guide for the manufacture and preparation of dosage forms, his use of these drugs at times would be chaotic and even dangerous.

In order to obtain an idea of which drugs were in use when THE JOURNAL-LANCET started publication in 1870, it might be wise to compare the present volume XV of the U.S.P. with those in use during the early life of this journal. The convention for revision to edit volume V was held in Washington on May 4, 1870. This book was published in 1875. For the next volume, the convention met on May 5, 1880, and volume VI was ordered published in 1882. Neither of these books records the presence of delegates from Minnesota. A delegate from Illinois was present at the convention for volume V of the U.S.P., and delegates from Illinois, Iowa, and Michigan were present at the convention for volume VI. No Minnesota delegates were appointed or attended these conventions until that for volume VII, which was held in 1890.

The following comparisons of the pharmacopoeias illustrate drug therapy then as compared with that at the present time. Volume V contained 383 pages; volume 6, 480 pages; and the present volume XV, 1,178 pages. A comparison of the contents is even more enlightening. The most common preparations in the old volumes were the extracts, solid or fluid. In volume V, 80 were included, and volume VI contained 120. Today's U.S.P. lists only a very few, of which liver, aspidium, parathyroid, and ipecac are important. Another common preparation was the tincture,

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of which volume V included 57, and volume VI listed over 70. The present U.S.P. lists only those of belladonna, iodine, and opium. Other preparations in the old pharmacopœias were ointments, 29 in volume V and 26 in volume VI; syrups, chiefly flavored, 22 in volume V and 34 in volume VI; and pills, generally extracts of crude plant drugs, 19 in volume V and 15 in volume VI. It is surprising that so few pills were in use at that time.

It is very evident that the drugs in use when THE JOURNAL-LANCET first began publication were chiefly of plant origin and of a crude preparation to be taken by mouth. Neither chemical analysis nor pharmacologic experimentation had begun to any great extent to isolate the active principles and to weed out the inactive substances.

Among pure drugs; some extracts from plants; and the remainder, synthetics, were the following in volume V of the U.S.P.: atropine, bismuth salts, bromides, calcium salts, chloroform, ether, morphine, mercury salts, alkaloidal extracts of Cinchona, silver salts, zinc salts, and iron salts. It is extremely interesting to note that, in almost every instance, the U.S.P. gave directions on how the drugs were manufactured. Morphine was made by extraction from opium, ether from alcohol, iron salts from iron wire, and so forth. Apparently, in 1870, no source was available from which to purchase these pure drugs. They had to be made by either a pharmacist or a physician.

In volume VI of the U.S.P. are to be found additional pure drugs, such as amyl nitrite, apomorphine, caffeine, chloral hydrate, codeine, quinine, quinidine, sodium salicylate, and a greater variety of metal salts. In this volume, methods of manufacture were omitted, suggesting that, by 1880, the manufacturing procedures had been taken over by concerns that are called pharmaceutical companies today. This revision of the U.S.P. showed a marked change and improvement as a whole, due largely to the part played by pharmacy in its revision.

The therapeutic benefits that a physician of 1870 to 1890 could give to his patients might be summarized as follows. For analgesia, morphine and, later, codeine were available. No one questions their efficacy. For minor analgesia, sodium salicylate was available. This drug, however, is quite unsatisfactory for most analgesic needs. Whether American physicians were using this drug in adequate doses in the treatment of acute rheumatic fever, no one knows. It had been introduced for this purpose ten years earlier in Europe. Aspirin was still unknown but was in-

troduced just before 1900; however, it did not become an official drug until 1926 in volume X of the U.S.P. The bromides and chloral hydrate were the only available hypnotics. The latter, especially, is still good enough to be used commonly today. With these two drugs, fairly satisfactory convulsive control could be maintained. There were no local anesthetics in the U.S.P., hence no local anesthesia. The beginnings of local anesthesia with cocaine were first described in 1884 by Karl Koller. For antisepsis and the local control infections, physicians had to depend on tincture of iodine, which produced adequate antiseptics of intact skin; mercury salts for local applications in the form of ointments or packs of aqueous solutions; silver salts in solutions; and, perhaps, also solutions of zinc salts. Today, with a wide variety of chemotherapeutic drugs available for the satisfactory control of most bacterial and protozoan infections, the foregoing armamentarium appears to be quite meager. However, when THE JOURNAL-LANCET was born, the beginnings of successful bacterial chemotherapy were still sixty years away. Only one hypotensive drug was included in volume VI of the U.S.P., namely, amyl nitrite. This drug and its over-all type of action were discovered by Sir Thomas Lauder Brunton in 1869. On the other hand, its dilator action on coronary arteries was not fully appreciated until after 1913. Quinine and quinidine were also included in volume VI. At that time, they were probably chiefly used in malaria, as antifever drugs in infections, and as uterine stimulants because the antifibrillatory action of quinine was not discovered until 1914 by Wenckebach and, of quinidine, until 1918 by Frey. Digitalis as a tincture, infusion, extract, and fluidextract was included in both volumes V and VI. It is interesting to speculate on what clinical effects physicians obtained with digitalis at that time. Although the drug had been introduced by William Withering before 1800, chiefly as a diuretic, its mechanism of action on the heart and its ultimate clinical usefulness were not appreciated until after the work of Cushny and others was published between 1895 and 1916. Again, it was shown after 1900 that extracts and fluidextracts were of doubtful activity compared to the tinctures. This was due to the development of the frog method of bio-assay of the digitalis glycosides, which was discovered, again, after 1900. Later, the cat method of assay was discovered, which then became official. From this method was obtained the cat unit and, later, the U.S.P. unit. With over 20 preparations of iron in the old pharmacopœias, the physicians of 1870 to 1885 certainly had at their disposal

a variety of drugs for the treatment of iron-deficiency anemia. How extensively these were used and whether anyone suspected that certain salts, such as ferrous sulfate, were more efficiently absorbed than certain other salts is unknown. Pointing to the inadequate use of iron salts at this time is the fact that it was not until after 1900 that the profession as a whole believed in inorganic iron absorption for the successful building up of hemoglobin.

Finally, it might be noted that these old pharmacopœias contained some 160 plant products as drugs, none of which are in use today or are known to have any significant pharmacologic actions. What, if any, might have been their use ninety years ago? Throughout most of our medical history, drug plants have been collected with great care at certain stages in their development and properly dried and stored in order to prevent putrefaction, mold contamination, and any spoilage from physical means. It is conceivable that many of these plants might have contained various vitamins, especially the water-soluble group. At a time when the American diet was not as varied or as complete as it is at present, these plants may have served a worthwhile purpose in helping to prevent serious vitamin deficiencies. From this standpoint, a number of inclusions in volume V of the U.S.P. are most interesting. This volume contained safflower, tapioca, prune, cod liver oil, olive oil, flaxseed oil, arrowroot, barley, oatmeal, starch, and marshmallow. These substances contain dietary substances which, at times, may offer therapeutic benefit—oils for various purposes; carbohydrates for easy digestion; gums for their demulcent action on the gastrointestinal tract; and, in at least one instance, vitamins A and D. Most of

these were deleted from volume VI of the U.S.P. Again, had suitable techniques for chemical analyses and pharmacologic manpower and experimental procedures been available, progress in drug therapy could have been more rapid.

By contrast, volume XV lists 123 tablets (pure drugs), 27 capsules, 112 injections, 65 solutions, and 13 vaccines and antitoxins as among the most important items included. This illustrates the effects of the chemist, whose analysis and isolation of drugs from organic sources together with the preparation of synthetic chemicals, which, with the work of the pharmacologist, have given us this large array of drugs with wide varieties of action. The large number of injections points to the significance of the hypodermic syringe in drug administration today.

CONCLUSION

The past ninety years have witnessed the most remarkable changes in drug therapy that the world has ever seen. Let us hope that the next century will produce just as remarkable an improvement in health. It can, if the efforts of medicine continue to be devoted to the ideals which have been prevalent since the time of Aesculapius.

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NICOTINIC ACID or tryptophan appears to protect rats against lethal doses of carbon tetrachloride. These agents stimulate the synthesis of pyridine nucleotides, presumably increasing the production of the mitochondrial coenzymes attacked by carbon tetrachloride. In this manner respiratory activity and tissue metabolism are maintained.

Seventeen test animals were given intraperitoneal injections of 50 mg. of nicotinic acid two days before and on the same day as administration of 0.6 cc. of carbon tetrachloride per 150 gm. body weight; 2 died as a result of the poisoning. In a later test, 5 of 16 animals given 50 mg. of tryptophan daily for three days died upon administration of 0.4 cc. of carbon tetrachloride.

C. H. GALLAGHER and R. A. SIMMONDS: Prophylaxis of poisoning by carbon tetrachloride. *Nature* Vol. 184, Suppl. 18, 1959, pp. 1407-1408.

The Emergence of Cardiac Surgery

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IMPRESSIVE PROGRESS has been made in recent years in the development of effective methods for surgical treatment of numerous afflictions of the heart. These advances, with but a few exceptions, have brought about remarkable changes in the outlook for almost every cardiac condition. Review of these developments from an historic point of view offers a particularly appropriate means of gaining a truer perspective of what has been accomplished. Moreover, as has been repeatedly demonstrated in virtually every field of human endeavor, the past is but a prologue to the future.

Ninety years ago, when the antecedent to THE JOURNAL-LANCET (The Medical and Surgical Journal) first appeared, no practicing physician or surgeon could have envisioned the need for such a résumé. The fact that the heart was out of the reach of the surgeon was certainly the universally accepted viewpoint of the day. However, the intervening years have been accompanied by medical and surgical achievements of such magnitude that they can truly be characterized as "performance beyond prophesy." This phrase was used by Dr. Owen H. Wangensteen¹ in a discussion of the progress which had been made in surgery on the occasion of the eightieth anniversary of the publication of this journal. The remarks made then, ten years ago, have been particularly prophetic of the advances that have occurred in the realm of cardiovascular surgery.

As we have pointed out previously,² this past decade has brought forth an impressive array of advances in the field of cardiac surgery. As recently as 1948, there were only three congenital cardiac lesions then amenable to surgical treatment: patent ductus arteriosus, coarctation of the aorta, and the systemic-pulmonary artery anastomotic procedure for tetralogy of Fallot. Prior to 1954, there was no patient alive who had

been cured of such relatively common cardiac malformations as a ventricular septal defect, atrioventricularis communis, or the lesions of tetralogy of Fallot. Even prior to 1956, such frequently encountered *acquired* cardiac lesions as mitral regurgitation and aortic insufficiency were beyond the realm of surgical correction. Today, these operations, as well as others not possible a few years ago, are regularly scheduled procedures.

Many factors have contributed to these gratifying advances. Perhaps most important in this regard has been the breaching of the barrier presented by the cardiac interior. In effect, the outer wall of the heart was the last anatomic frontier of the many that have confronted surgeons through the ages, and, as such, it represented an important obstacle to progress.

The development and introduction into routine clinical use of safe and effective methods of extracorporeal circulation for cardiopulmonary bypass has removed this barrier and opened a new era in the therapy of cardiovascular disease. A steadily increasing variety of progressively disabling cardiac lesions, both congenital and acquired in origin, and in many cases previously inoperable, have been successfully treated under direct vision by means of open cardiotomy utilizing extracorporeal circulation. In many cases, these methods have allowed complete correction of cardiac lesions previously considered incurable. In others, these direct-vision techniques have resulted in improved and safer surgical treatment for lesions for which less satisfactory means were available.

BEGINNINGS OF CARDIAC SURGERY

The early history of surgery was intimately linked to society's increasing need for a means of treating the consequences of trauma, and the surgeon evolved, in a large part, as a response to this need for emergency assistance. When primitive man emerging from his cave first experienced a pang of compassion for his wounded neighbor and helped nature staunch the flow of

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blood, cardiovascular surgery was born. The march of progress during some centuries was slow, but, undeniably, Antyllus, Paré, Cesalpino, Harvey, John Hunter, Astley Cooper, Lister, Carrel, Landsteiner, and Fleming among many others laid the foundations for the phenomenal advances of the past few years.

Subsequently, as many of the technics and principles involved in the treatment of wounds were mastered, the lessons learned and the experiences acquired permitted attacks upon other less acute problems; thus elective surgery came into being. Therefore, it seems proper to consider initially the contributions of those who were "the first" to operate upon the heart in an attempt to repair the wounds it sustained. Their successes served as beacons to those who followed and demonstrated that even this most vital organ could respond favorably to the ministrations of the surgeons. But they can hardly fall heir to the fame and glory of having been "the first" without deferring much to their predecessors—those who created surgery as we know it today and placed it upon the firm foundation of scientific study which has sustained it throughout its recent rapid and fruitful growth.

Among the greatest of the predecessors of cardiovascular surgeons was John Hunter, one of the first very great men in the history of surgery. He was born in 1728 in Scotland and died suddenly of a coronary thrombosis on October 16, 1793, in London at St. George's Hospital. John Hunter fell lifeless during the course of a heated argument with his colleagues and the board of governors of St. George's Hospital concerning differences of opinion over revision of the teaching curriculum of the medical school and division of private income—fees paid to the teachers by medical students of that day³—two subjects still not unlikely to produce passionate outbursts of emotion in medical schools today. He dominated the medical life of the eighteenth century. He wrought mighty changes in all fields by combining effectively for the first time the study of anatomy, pathology, and physiology together with the practice of surgery. All of this he leavened with indefatigable energy. His significance to cardiac surgery in particular is the fact that he was the first exponent of scientific research in the modern sense. It is the application of these very same methods that has resulted in the present advances in cardiovascular surgery.

In an historic paper written in 1926, Claude Beck⁴ reviewed the history of wounds of the heart. A more recent and extensive review of

cardiac surgery by George H. A. Clowes⁵ also has an excellent discussion of this topic. Paget's⁶ book, *The Surgery of the Chest*, cites a surgeon by the name of Romero as having been one of the earliest to approach the area of the heart surgically. In 1819, he opened the pericardium for effusion in 3 patients; 2 survived. Some ten years later, Larrey, Napoleon's surgeon, successfully decompressed a wounded heart with a trocar after performing a thoracotomy. In 1868, Georg Fischer⁷ compiled a series of 452 wounds of the heart in both human beings and animals, none of which was treated surgically. He reported a recovery rate of only 10 per cent. However, despite the frequency of this lesion and its lethal consequences, there had been no recorded attempt to suture a wound of the heart. Experimental work in this area was inaugurated by Block⁸ in 1882. In one of the earliest experiments in cardiac surgery, he repaired wounds made in the hearts of rabbits and showed that an animal could recover from such a procedure. The feasibility of applying this to the human heart was suggested. However, in 1883, none other than so prominent a surgeon as Billroth is reputed to have said that "The surgeon who should attempt to suture a wound of the heart would lose the respect of his colleagues."¹ It is indeed fortunate that this admonition was not taken too seriously in later years.

In 1893, an American surgeon, Daniel Hale Williams,⁹ operated upon a patient to repair a puncture wound of the heart (figure 1). Dr. Williams was the first man to undertake such a procedure. He also was the first outstanding and nationally known surgeon of the Negro race in America (figure 2). Unfortunately, his contribution has often been overlooked, and he has not generally received the recognition he deserves for it. It has been said of him in this regard, "Like Solomon, he was black but comely, singing but unsung."¹⁰ The operation took place at the Provident Hospital in Chicago under the following circumstances:

"James Cornish, an expressman, age twenty-four years, during an altercation received a stab wound through the fifth costal cartilage, injuring the internal mammary vessels and wounding the pericardium and heart. . . ." He continued to bleed and at operation hours later, "The original wound was lengthened to the right as far as the middle of the sternum. . . The sternum, cartilage, and about one inch of the rib were exposed. . . The incised piece of chest wall was reflected upward, making an opening about two inches long and one and one-half

STAB WOUND OF THE HEART AND PERICARDIUM—SUTURE OF THE PERICARDIUM—RECOVERY—PATIENT ALIVE THREE YEARS AFTERWARD.

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The following case is deemed of sufficient importance to be reported three and a half years after its occurrence. The writer's illness about the time of the patient's recovery and a change of duties to his present position have prevented previous publication.

James Cornish, an expressman, aged twenty-four years, during an altercation received a stab wound through the fifth costal cartilage, injuring the internal mammary vessels and wounding the pericardium and heart. The kind of knife and length of blade could not be determined from the patient.

He was admitted to Provident Hospital, Chicago, July 9, 1893, at 7:30 P.M., with a stab wound about one inch long, three-fourths of an inch to the left of the sternum, through and in the long axis of the fifth cartilage. A probe recognized only a superficial wound, but during the night there were such persistent



FIG. 1.—James Cornish, Aged Twenty-four

hemorrhage, pain over the cardiac area, short, sharp, chill cough, and such pronounced symptoms of shock that a re-examination the following morning showed

Fig. 1. First recorded attempt to surgically repair a wound of the heart, 1893. The heart did not require sutures; the lacerated pericardium did."

inches wide, bringing the internal mammary vessels into view. These vessels were ligated above and below with small catgut. . . . The heart and lung being depressed backward, a small punctured wound of the heart, about one-tenth of an inch in length and about one-half



Fig. 2. Daniel H. Williams, the Negro surgeon who first exposed the heart in an attempt to suture a laceration of it. He was one of the founders of Provident Hospital, Chicago, and a charter member of the American College of Surgeons.¹⁰

of an inch to the right of the right coronary artery between two of its lateral branches, was seen. The wound in the pericardium was about one and one-fourth inches in length. There was no hemorrhage from the heart or pericardium. The edges of the pericardium were held by a long smooth forceps and a continuous suture of fine catgut was made. . . ."

Although Dr. Williams only performed a pericardiorrhaphy, his was the first recorded successful attempt at operative correction of a cardiac wound. The report appeared in 1897, three and one-half years after its occurrence.

Meanwhile, in 1895, Del Vecchio¹¹ had reported his dog experiments, demonstrating healed wounds of the heart to the eleventh International Medical Congress in Rome. Later that same year, Capellen¹² in Norway sutured a wound of the heart, but the patient died on the third postoperative day. Farina of Rome, in March 1896, also failed in an attempt to suture a wound of the human heart. Six months later, Louis Rehn¹³ of Frankfurt achieved the first clear-cut success. A one-half inch wound in the right ventricle was closed and the patient survived.

In this country, interest in wounds of the

heart was demonstrated by Dr. Luther L. Hill of Alabama. In 1900, he reviewed 17 cases of successfully sutured wounds of the heart.¹⁴ The subsequent achievements of Dr. Hill¹⁵ were most aptly described by his son, Senator Lister Hill of Alabama, on the occasion of the thirteenth annual meeting of the Society for Vascular Surgery. Senator Hill was presented with a plaque commemorating the pioneering contributions to cardiovascular surgery of his father and said:

"In 1902 came the opportunity for which he (Dr. Hill) had been eagerly waiting. In September 14th of that year he successfully sutured the heart of a 13-year-old Negro boy where the cavity of the heart had been penetrated by the blade of a knife. May I remind you, my friends, that this, the first successful suture in these United States, was performed by the flickering light of two kerosene lamps on an old kitchen table in a Negro shack. In the *Medical Record* of November 29, 1902, my father reported the case."¹⁶

Ten years after he reported the first successful closure of a wound of the heart, Rehn reported upon 124 cases, a collected series of his and other surgeons' experience, with a 40 per cent recovery rate. By 1920, Tuffier could boast of a 50 per cent recovery rate. Thus did surgeons unequivocally demonstrate for the first time that operative procedures upon the heart were not only possible but were at times lifesaving.

ADVANCES WHICH CONTRIBUTED TO THE DEVELOPMENT OF CARDIAC SURGERY

After the initial successes with traumatic wounds, further progress in the operative treatment of diseases of the heart was slow and tedious. Aseptic and antiseptic techniques were in their infancy. Anesthesiology was a fledgling branch of medicine which employed crude and inefficient methods. A gross deficiency existed in the surgeon's understanding of the physiologic responses to major surgery and the normal and abnormal physiology of the respiratory and cardiovascular systems, not to mention the large gaps in knowledge in such basic areas as body fluid and electrolyte balance. It was only after these many basic but vital problems were to some degree solved that further advances could be made.

One of the outstanding contributions consisted of the development of techniques for maintaining ventilation and anesthesia with an open chest. Thoracic surgery required the competent mastery of this problem before it could hope to be productive. *The Principles of Thoracic Anaes-*

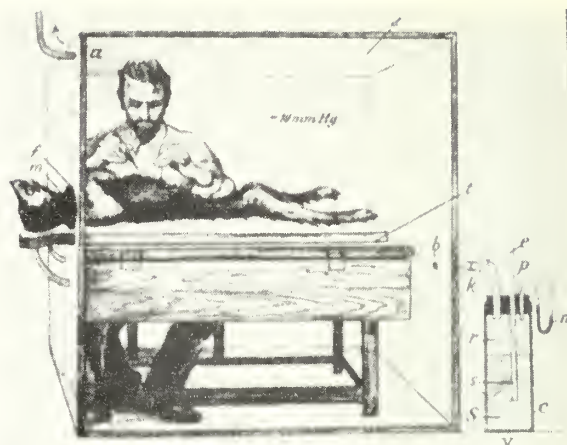


Fig. 3. A negative pressure operating chamber devised by Sauerbruch in 1904 for use in experimental open chest operations on dogs. Note that the head of the animal was kept outside of the chamber.¹⁷

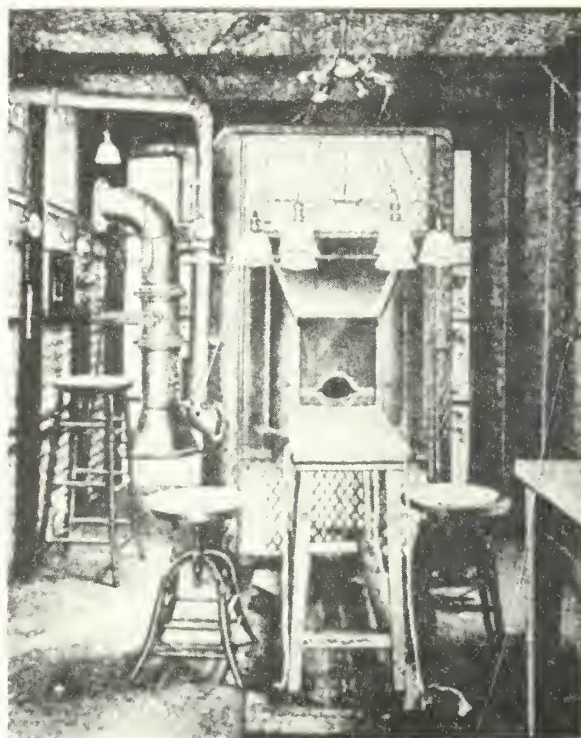


Fig. 4. Interior of Meyer's negative pressure chamber. Note operating table and opening in the operating chamber through which the patient's head protruded.¹⁷

*thesia Past and Present*¹⁷ by Mushin and Rendell-Baker may be read for a full exposition of how the "pneumothorax problem" was finally resolved. Progress along these lines was not continuous and straightforward. Other devoted and competent investigators diverted attention to techniques which ended in failure, although they contributed to general knowledge. The neg-

ative operative operating chambers of Sauerbrueh (figure 3) and of Meyer (figure 4) are examples of this. Their efforts, while laudable in their aim to overcome the physiologic barrier presented by the intact thorax, did perhaps delay the universal acceptance of elective thoracic surgery by their vehement opposition to a simple physiologic solution to this problem. They ardently opposed the concept of maintaining ventilation and anesthesia by means of controlled positive pressure administered through an endotracheal tube. The intrinsic lessons provided by this controversy provided valuable orientation to us in some of the developmental phases of open heart techniques.¹⁸ It was a guiding principle in developing the concept that simple techniques would ultimately prevail in heart surgery as they had previously in lung surgery.

In 1904, Sauerbrueh¹⁹ presented the results of eleven years of experimental work with the "pneumothorax syndrome." He drew attention to the need for maintaining a differential pressure within and without the lung. By 1910, the complexity of operating in an airtight chamber, which contained the whole surgical team as well as part of the patient, was abandoned. Attention was then focused on the use of positive pressure devices. Some of these also assumed rather im-

practical proportions (figure 5). In 1909, Meltzer and Auer²⁰ proposed a very simple method for positive pressure anesthesia, which met with a great deal of opposition at the time of its introduction. However, in an era dominated by dogmatic views, they steadfastly stood by their experimental work and defended their conclusions on that basis. Their method utilized intubation of the trachea and did not depend upon the respiratory movements of the patient. It is particularly revealing that their method, although extremely important, was far from original. In the second edition of *De Humani Corporis Fabrica* (1555), Vesalius described the technic of transtracheal intubation, using a tube of reed or cane, which he employed while studying the anatomy of living animals.

Despite the opposition of some of the most prominent thoracic surgeons during this period, others were eager to take advantage of any successful method of maintaining a patient during thoracic surgery. Elsberg²¹ lost no time in adopting Meltzer's work and, by 1910, had introduced the use of an anesthesia machine and a laryngoscope to effect endotracheal intubation. After some of the deleterious effects of carbon dioxide retention were better understood and further refinements in anesthetic techniques occurred, open chest surgery became a feasible undertaking. It should be emphasized that this brief description of the mastery of the "pneumothorax problem" does not do justice to the impressive number of imposing personalities who were involved in this dramatic phase of the development of thoracic surgery. One may consult the reviews of Graham²² and Blades²³ for a more complete account of the evolution of this area of surgical endeavor.

Landsteiner's work with the blood groups had also taken place during the early part of the twentieth century, and, by 1915, Richard Lewishahn²⁴ of New York had instituted the use of blood transfusions by adding a sodium citrate solution to freshly drawn blood. Here, too, it is remarkable how long opposition and criticism delayed widespread acceptance²⁵ of this basic technic. This is yet another important lesson to the investigator who seeks to change established tradition: Do not be discouraged by opposition, even if vehement.

It was not until the ravages of World War I and, later, World War II that the problem of shock received the intense study it deserved. It was indeed surprising, at least in retrospect, that, when a patient had obviously lost blood in large quantities, the fact that he needed blood

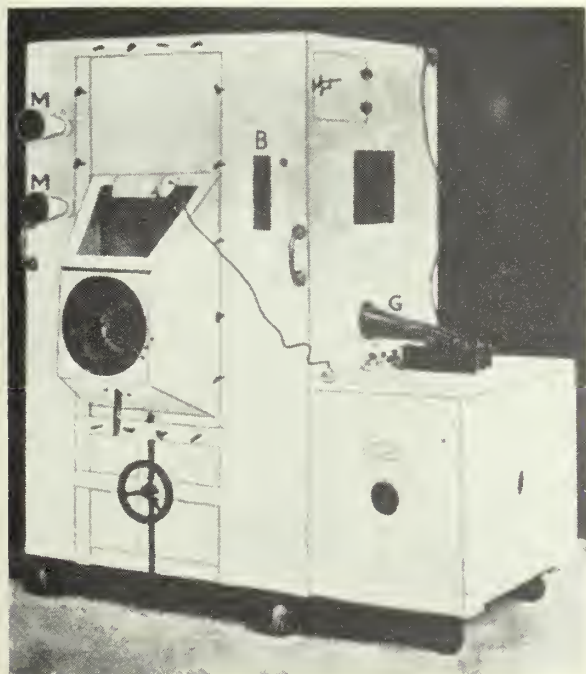


Fig. 5. A positive pressure apparatus into which the patient's head was placed. Anesthetist also sat in this chamber.¹⁷

for replacement was so slow in receiving general acceptance. Although many questions concerning shock still remain unanswered, the hazards of hypovolemic hypotension and the need to control blood loss gradually became well-learned lessons. An understanding of the fluid and electrolyte requirements of the surgical patient also increased through the years, serving to make major operative procedures safer for the patient. It is also obvious that the introduction of antibiotics has given an added measure of protection to the patient who must undergo extensive surgery.

It would be a serious omission if mention were not made of the contributions of the many physiologists and cardiologists whose efforts have resulted in unfolding to some degree the complex dynamics of the cardiovascular system. The surgical correction of various congenital and acquired cardiac defects was also very much dependent on the refinements in diagnosis which internists and radiologists and surgeons have made, partially as a response to surgical requirements.

Throughout these years of great progress in surgery, when operations on the heart could not yet be considered a feasible undertaking, there were many prophetic and impatient men who yearned to see them done. As early as 1902, Sir Lauder Brunton²⁶ stated that "the good results that have been obtained by surgical treatment of wounds of the heart emboldens one to hope that before long similar good results may be obtained in cases of mitral stenosis." Five years later, in 1907, John Munro²⁷ suggested that the congenital defect of a patent ductus arteriosus could be corrected by surgical means. In 1912, Tuffier²⁸ tried to stretch a stenosed aortic valve by introducing his finger through the invaginated wall of the aorta, a dubious technic but nonetheless worthy in its goal. Doyen's²⁹ attempt to section a stenosed mitral valve was followed in 1913 by an attempt to relieve a pulmonic stenosis. In this country, Cutler and associates³⁰ reported on their disappointing experiences with the surgical treatment of mitral stenosis. Two years later, in 1925, Souttar³¹ of London undertook to dilate a stenosed mitral valve by inserting his finger through the atrial appendage in much the same manner as is done today. Souttar's patient survived and seemed to improve, but the procedure was not accepted as safe and beneficial. In a letter to Brian Blades,²³ Souttar explains why he was unable to obtain another case: "At that time, however, it was an article of faith with physicians that the

valves were of no importance and that the only thing that mattered was the condition of the cardiac muscle. In that atmosphere, I was naturally unable to obtain another case in spite of this one complete success. It was a pity but easy to understand." Perhaps, had he been more persistent in attempting to extend his success by repetition, cardiac surgery might have been pushed ahead immeasurably. His operation, unlike the one devised by Cutler and Levine,³² did not entail the use of a cardioscope. Souttar's technic for correction of mitral stenosis was reintroduced over twenty years later by Bailey.³³ In 1937, John Strieder of Boston ligated a patent ductus arteriosus in a patient with active bacterial endocarditis. The patient died on the fourth postoperative day. Fifteen months later, Robert Gross achieved world-wide recognition for successfully curing a child with this congenital deformity. This achievement can be looked upon as a milestone in the development of heart surgery, for this was the closest juxtacardiac lesion that had been to that date effectively and safely treated by operative means. Meade's³⁴ historical review of the development of surgery for patent ductus arteriosus may be read by those interested in this phase of the history of cardiac surgery.

ADVANCES WHICH CONTRIBUTED TO THE DEVELOPMENT OF INTRACARDIAC SURGERY

It was clear that thoracic surgery was becoming more adventurous and that cardiac surgeons were advancing steadily toward their goal—the interior of the heart itself. In 1939, O'Shaughnessy³⁵ predicted that "the real key to further advance in the surgical treatment of established cardiac defects will be provided by the provision of some simple and efficient method of maintaining cerebral circulation while the heart is out of commission." It is of interest to note O'Shaughnessy's emphasis upon "simple and efficient" in his prediction, since ultimately the first successful methods for open heart surgery—hypothermia for atrial defects and cross circulation for ventricular defects—both met those requirements admirably. Had O'Shaughnessy not come to an untimely death in the Armed Forces during World War II, he may well have made important contributions to the development of this field.

There was still much to be accomplished, however, for juxtacardiac lesions. In 1944, a coarctation of the aorta was successfully resected by Gross and Hufnagel³⁶ and Crafoord and Nylin³⁷ independently and at about the

same time. That same year Blalock and Tausig³⁸ introduced the systemic-pulmonary artery anastomotic procedure for palliation of the tetralogy of Fallot malformation. A more vigorous surgical approach to the acquired cardiac lesions was also becoming evident at this time. The early attempts which were made to correct valvular deformities have been mentioned and will be dealt with again later.

Much interest also centered about the possibility of increasing coronary artery blood flow. The first studies in this area were initiated in 1932 by Dr. Claude Beck. The operation of cardiac decortication, suggested as early as 1895 for constrictive pericarditis, also underwent improvements during this period.

Encouraged by these events, efforts directed at the surgical correction of mitral stenosis were launched by Bailey³³ and Harken and associates.³⁹ Harken's⁴⁰ success during World War II with the surgical removal of foreign bodies from the chambers of the heart undoubtedly stimulated his interests in cardiac surgery. After Sellors⁴¹ and, later, Brock⁴² demonstrated that pulmonary stenosis could also be successfully treated by operative means, it became evident that the walls of the heart had to be opened if further advances were to be achieved.

In order to perform visual intracardiac surgery, a bloodless field was required. Several different methods had been considered and found useful. These fell into one of two categories: cardiopulmonary bypass or hypothermia. Although the former had received a greater amount of attention and had seemed to show greater promise, hypothermia will be discussed first, since it was with this technic that the world's first successful intracardiac operation under direct vision was performed.

Hypothermia. In 1950, Bigelow and associates⁴⁴ published their experimental results with general body hypothermia. Their experience had been accumulated over a two-year period, during which time 120 dogs were cooled and rewarmed. The express purpose of these experiments was to provide "a simple procedure allowing operations on a diseased or abnormal heart." Earlier that same year, they described the use of general hypothermia for experimental intracardiac surgery.⁴⁵ The opening paragraph of this paper, which was presented at the 1950 Meeting of the American Surgical Association, reads as follows: "General hypothermia is being investigated as a means of reducing the oxygen requirements of the body sufficiently to allow exclusion of the heart from the circulation,

thereby permitting intracardiac surgery under direct-vision." This latter paper is outstanding in many respects. It also describes the use of an artificial pacemaker which provided periodic electrical stimulation to the sinoauricular node.

The following year Bocrema of Holland and his associates⁴⁶ related their experiences with this technic and found that they had time, "to perform à vue an intracardiac operation." However, it remained for Dr. F. John Lewis and Dr. Richard L. Varco of the University of Minnesota Hospitals to perform the first successful intracardiac procedure under direct vision.⁴³ Working with Mansur Taufic, they had perfected the technic in the experimental laboratory. When they were satisfied "... that the method had reached the stage where clinical trial was justified, Dr. Varco kindly sought and found a suitable candidate" and assisted Dr. Lewis with the operation. This patient was a 5-year-old girl who had been sickly and underdeveloped as a result of a congenital atrial septal defect. The operation took place on September 2, 1952, at the University of Minnesota Hospitals. Cardiac inflow was occluded for five and one-half minutes. During this period, the 2 cm. defect in the secundum area was closed in the open empty right atrium. Her recovery was uneventful.

General body hypothermia has been used extensively by many surgeons since this report appeared, and much has been accomplished using this technic in the repair of simple atrial (secundum) defects and valvular pulmonary stenosis. Its ultimate place in cardiac surgery as a primary modality, however, is still quite uncertain due to the rapid development of bypass equipment and technics. It may be of greater value now as an adjunctive measure during operations on the heart. Selective cardiac hypothermia⁴⁷ combined with cardiopulmonary bypass—a combination of normal systemic blood temperature with the heart cooled 10 to 20° C. by coronary perfusion of cold blood—allows the surgeon a motionless operative field without the dangers of the myocardial hypoxia which characterizes the other types of induced cardiac arrest. Also, general hypothermia has been utilized in conjunction with the pump-oxygenator to reduce flow requirements.⁴⁸ The ultimate role of this combination likewise remains to be defined at this time.

Cardiopulmonary bypass technics. In 1937, John H. Gibbon, Jr.,⁴⁹ described his experience with the use of an artificial means of maintaining circulation during experimental occlusion of the pulmonary artery (figure 6). His pioneering

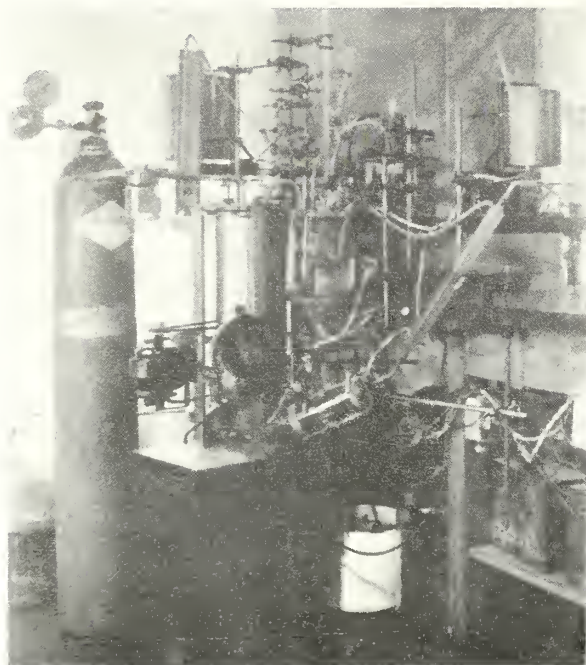


Fig. 6. First oxygenator used for cardiopulmonary bypass in animals, which was devised by John H. Gibbon, Jr., in 1937."

work initially started while he was working with Dr. E. Churchill in the Surgical Research Laboratories at the Massachusetts General Hospital. Their interest arose out of a gross dissatisfaction with pulmonary embolectomy as described by Trendelenburg. They felt that a great benefit would be accrued if the heart and lungs could be bypassed while the operation was being performed. Gibbon wrote:

The object of these experiments was to determine whether the circulation could be aided by artificial means in the presence of partial or complete occlusion of the pulmonary artery. The means employed were the withdrawal of blood from a peripheral vein, the introduction of oxygen into that blood, and the reinjection of the blood into a peripheral artery in a central direction. The blood was thus short circuited around the obstruction in the pulmonary artery and a good part or all of the work of the heart and lungs was temporarily taken over by artificial means.

Earlier in the paper, he stated that, "So far as I am aware, there has been no report of a successful temporary substitution of an entirely mechanical apparatus for the heart and lungs of an animal." There had, however, been a number of successful prolonged perfusions of isolated organs. In 1931, Staub⁵⁰ had described a screen-type oxygenator for use in isolated liver perfusions *in situ*, which was virtually identical with the oxygenator subsequently adopted by

Gibbon.^{51,52} Gibbon continued to work in this field after he left Boston for Philadelphia to become professor of surgery at the Jefferson Medical College.

A group of Scandinavian workers, which included Crafoord, Anderson, and Bjork, had been working along similar lines. Crafoord's experience with operations for patent ductus arteriosus prompted him to attempt to devise a bloodless approach to the inside of the heart, arresting the circulation of blood but maintaining an adequate flow through the brain with a cardiopulmonary machine. In 1946, Crafoord and Anderson constructed an artificial heart which Bjork⁵³ used in experiments on animals.

In 1951, a year after Bigelow had presented his work with hypothermia to the American Surgical Association, Bernard J. Miller and Dr. and Mrs. Gibbon⁵¹ presented a paper to the same group on "Recent Advances in the Development of a Mechanical Heart and Lung Apparatus." Their machine consisted of a battery-type screen lung which utilized the roller-type pumps described by DeBakey in 1934. On that same program was a paper from the University of Minnesota Hospitals. The senior author, Dr. Clarence Dennis,⁵⁴ having spent the previous four years developing a pump-oxygenator, reported an unsuccessful operation upon a patient for repair of an atrial septal defect. Cardiopulmonary bypass was effected by an apparatus which consisted of a screen oxygenator and

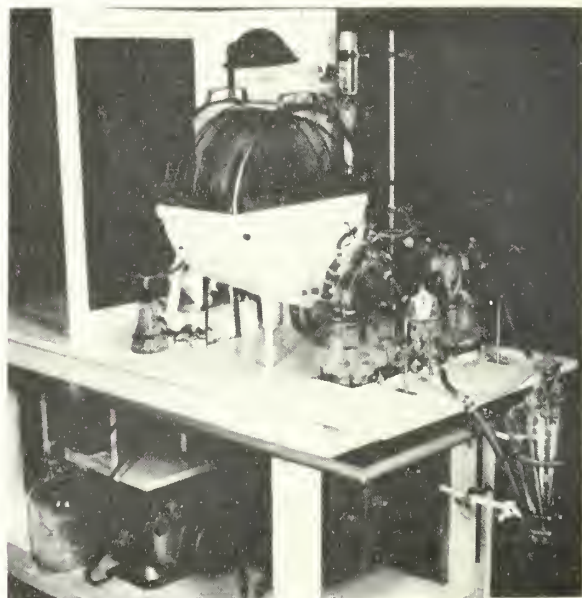


Fig. 7. Pump-oxygenator apparatus devised by Dennis and associates⁵⁴ in 1951 for cardiopulmonary bypass procedures.

modified Dale-Shuster pumps (figure 7). The latter, a type of double perfusion pump, had been described in 1928. The following year, Gibbon also operated unsuccessfully upon a patient using his mechanical heart-lung, and it was not until May 1953 that the first successful intracardiac procedure under direct vision using cardiopulmonary bypass for closure of an atrial secundum defect was performed.⁵² This milestone in the history of cardiac surgery is a tribute to the perseverance and ability of John H. Gibbon, Jr.

However, the apparatus and techniques for total cardiopulmonary bypass were all very complex and did not lend themselves readily to widespread or even repeated use. Atrial septal defects were already being closed under direct vision routinely with good results at that time at the University of Minnesota Hospitals and several other centers utilizing general body hypothermia. Attempts by Gibbon and others to repeat the successful closure of an atrial septal defect utilizing the heart-lung machine or to extend this technic to other more complex lesions within the ventricles failed. In several of these unsuccessful operations, the pump-oxygenator seemed to function smoothly, and the surgical procedure itself appeared to be technically successful; yet the patient failed to survive and the cause of death remained obscure. This occurrence, repeated several times in the hands of different workers, gave rise to the opinion, then prevalent, that the fault was not in the methods but rather that the problem was the "sick" human heart. The latter, damaged by the pathology which the surgeon sought to correct by surgical means, could not be expected to tolerate a cardiotomy, especially into a ventricle, together with an extensive intracardiac procedure. It was therefore felt that this field of open intracardiac surgery was doomed to remain one of very limited application. It was at this point that controlled cross circulation⁵⁵ appeared upon the scene (figure 8). After extensive experimental investigations on March 26, 1954, the first of 45 patients, all with complex types of intracardiac pathology, was operated upon (figure 9). For the first time, such relatively common congenital intracardiac lesions as ventricular septal defects, atrioventricularis communis, and tetralogy of Fallot were completely corrected by open cardiotomy.^{18,55,56-62} Indeed, in the course of a few months, the prevalent pessimism in regard to the potential applicability and feasibility of intracardiac surgery previously mentioned was completely changed, for 19 of the first 27 patients with ventricular septal defects operated

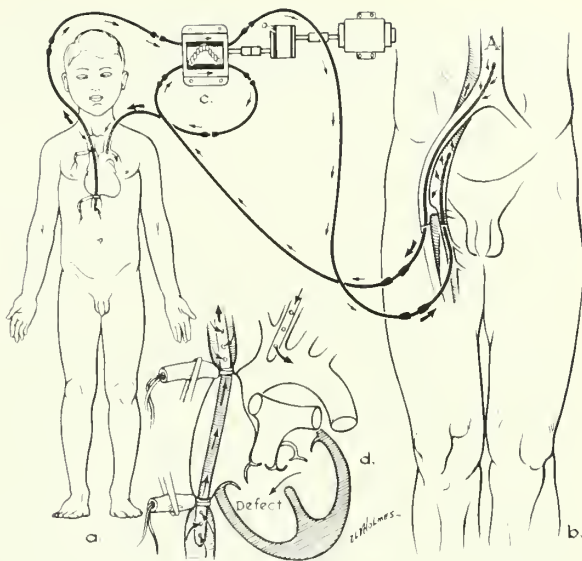


Fig. 8. Schematic depiction of cross circulation procedure used to effect cardiopulmonary bypass. The simple pump between patient and compatible donor served to control the interchange of arterial and venous blood.

upon survived.⁶² All of these were, as we now recognize, patients with advanced disabilities due to pulmonary hypertension, and many were infants in chronic cardiac decompensation. Likewise, it was possible to successfully achieve complete intracardiac correction, with survival in 6 of the first 10 patients operated upon with the tetralogy of Fallot malformations.⁵⁹ Finally, 1 infant with a complete form of the atrioventricularis communis defect and severe pulmonary hypertension was completely corrected in 1954 with prompt regression of the pulmonary pressures to normal as demonstrated by postoperative recatheterization.⁶²

These accomplishments were made possible by the simplicity and physiologic efficiency of the cross circulation method, supplying as it did perfectly oxygenated blood to the patient,⁶³ together with the application of the "low flow" or "azygos flow" principle,^{64,65} which permitted the use of low perfusion rates of the patient without deleterious effects.⁵⁵ Although there had been no mortality among the donors and no serious complications in those utilized as donors, methods which did not require another human lung were sought to effect extracorporeal oxygenation now that it was apparent that not only were these operations possible but actually could be performed with gratifying results in very sick patients. Several new techniques, such as the use of a membrane oxygenator^{66,67} (heterologous dog lung) and a reservoir of arterialized venous blood,^{68,69} were briefly employed but

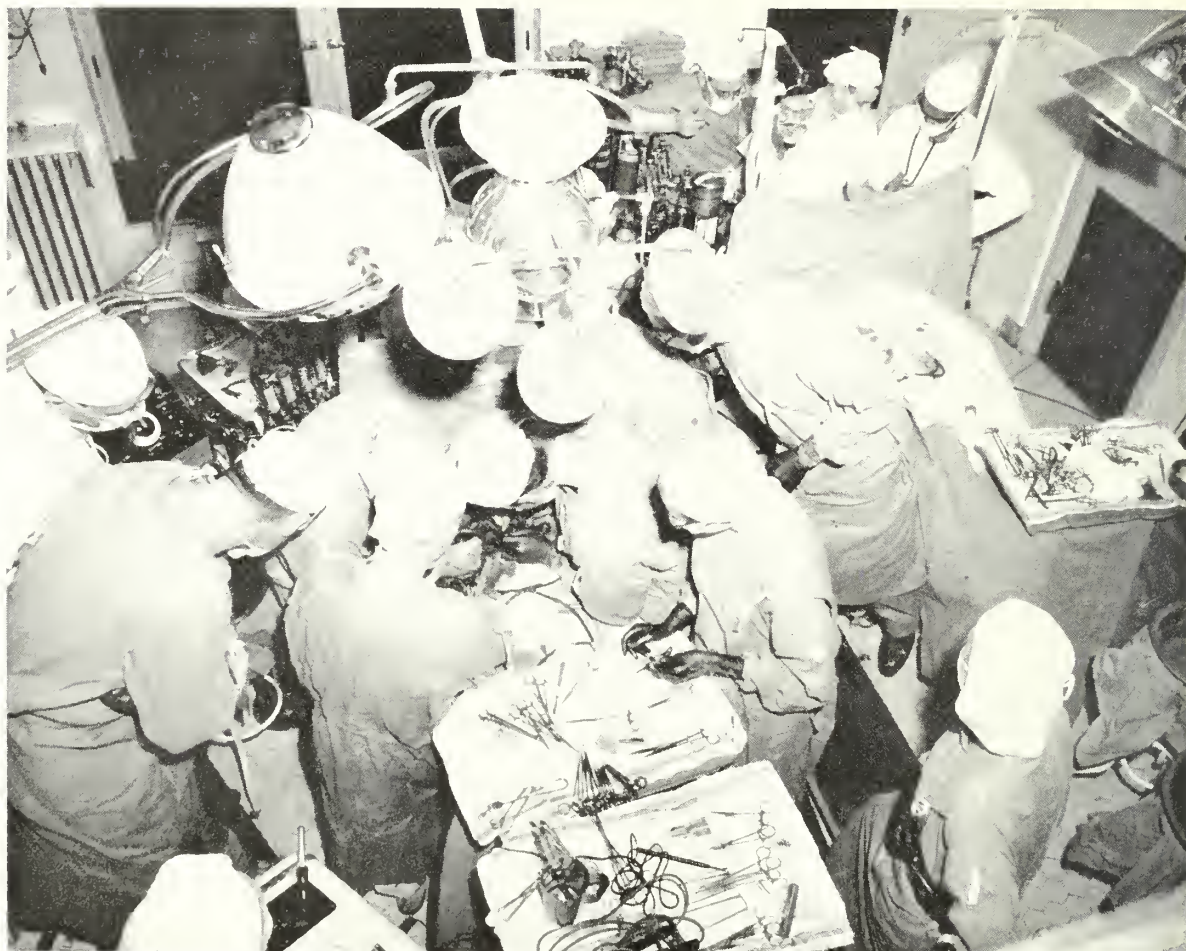


Fig. 9. Photograph taken in 1954 in the operating suite at the University of Minnesota Hospitals during the operative procedure resulting in the first successful correction of a ventricular septal defect. The technic utilized was total cardiopulmonary bypass by the method of controlled cross circulation (figure 8). The donor, serving as the oxygenator, may be seen at the far right with cannulae inserted into the common femoral artery and saphenous vein, respectively. The Signamotor pump controlling both the arteriovenous interchange and serving also to replace the function of the patient's bypassed heart is situated between donor and patient and is obscured in this photograph. At the moment of this photograph, the patient's heart is open and silk stitches are being placed in the margins of the ventricular septal defect.

ultimately were superseded by perfection of completely artificial pump-oxygenators. Experimental work with a bubble oxygenator by Dr. R. DeWall soon bore fruit, and by 1955 it was in routine use^{70,71} (figure 10). Its efficacy as a means of bypassing the heart and lungs has been proved in the course of over 1,000 open operations at this clinic.

ACQUIRED HEART DISEASE TREATED BY CARDIOPULMONARY BYPASS TECHNIQS

Attention next turned to acquired heart lesions. In early 1955, a patient with an acquired traumatic ventricular defect was successfully treated at the University of Minnesota Hospitals utiliz-

ing the heterologous (dog) lung for extracorporeal oxygenation.⁷² This first successful application of total cardiopulmonary bypass to acquired heart disease stimulated our interest in the many other forms.

By late 1955, the rapidly increasing experience with the use of the pump-oxygenator also resulted in initiation of experimental work⁷³ upon possible techniques for repair, under direct vision of other acquired valvular cardiac conditions.

These studies soon bore fruit, and, in 1956, for the first time, successful direct vision reparative operations utilizing cardiopulmonary bypass were carried out for calcific aortic stenosis,⁷⁴ mitral insufficiency,^{75,76} aortic insufficiency,⁷⁷ and

mitral stenosis.⁷⁷ By October 1958, the technic of direct coronary perfusion to achieve selective cardiac hypothermia and the pump-oxygenator had been perfected to a degree which permitted successful resection of the aortic valve with placement of a subcoronary valvular prosthesis.⁷⁸

Currently, there is widespread interest in the development of both surgical methods and prostheses for correction of all types of acquired valvular abnormalities. It is quite predictable that this field of open heart correction for acquired cardiac disease will continue to grow at a rapid rate, for two important reasons. First, the increased accuracy and scope which visual technics give to the surgeon have increased the effectiveness of surgical treatment for such conditions as aortic stenosis and complicated types of mitral stenosis formerly treated by closed or blind methods. Second, for other conditions, such as mitral insufficiency or aortic insufficiency, it is possible to offer for the first time in the long history of acquired valvular heart disease a definitively corrective operation for the patient with advancing disability.

UNIVERSITY OF MINNESOTA VARIETY CLUB HEART HOSPITAL

In 1945, in an entirely different and seemingly unrelated environment, an idea was born which undoubtedly exerted considerable influence in the accelerated development of heart surgery; in particular, open heart surgery. This idea, to build a hospital devoted entirely to heart disease, was fostered initially in the minds of a small group of men who were members of the Variety Club of the Northwest, a service organization made up of members from all walks of the show and entertainment business— theatre owners and managers; stage hands; radio, television, stage, and screen performers; theatre attorneys; film distributors, and so forth. These imaginative lay pioneers of the frontiers of medical progress were concerned with the appalling disabilities and deaths due to heart disease. As a consequence, they conceived the idea of doing something about the problem by raising a sufficient sum of money to build a Heart Hospital and present this hospital to the University of Minnesota Medical School in Minneapolis. There was no precedent for such a project, and, as so often happens when completely new ideas are presented, considerable opposition was encountered, even among some members of the Medical School staff directly concerned with diagnosis and treatment of heart disease. They argued that it was totally illogical to build an entire hospital



Fig. 10. Operating room scene in the University of Minnesota Hospitals in 1955, during which the disposable plastic bubble oxygenator is being used to effect total cardiopulmonary bypass.

devoted to diseases of a single organ of the body. However persuasive this argument may have been, logic and great achievements have never been constant handmaids, except perhaps in retrospect, and fortunately these lay humanitarians were undeterred by their opposition. In 1951, the Variety Club Heart Hospital with 80 patient beds and diagnostic and research laboratories (figure 11) opened its doors to become the first such hospital in medical history. It would, indeed, be difficult to overestimate the impact of the availability of these experimental and clinical facilities upon the rapid developments that have followed.

SUMMARY

An attempt has been made to indicate the manner in which cardiac surgery has emerged as a means of treating both acquired and congenital cardiac lesions. In common with the majority of surgical undertakings, it first evolved as a response to man's need for the treatment of trauma. After much travail, the basic elements in the body's response to surgery, infection, anesthesia, blood loss, and the open chest were better understood. This emboldened surgeons to



Fig. 11. The University of Minnesota Variety Club Heart Hospital—the first hospital in the world devoted entirely to diagnosis, study, and treatment of heart disease. It was at this hospital, located along the Mississippi River, that open heart surgery was born. The first patients in medical history to have successful intracardiac corrective surgery under direct vision of such varied and manifold lesions as atrial septal defect, atrioventricularis communis, ventricular septal defect, tetralogy of Fallot, ruptured aneurysm of sinus of Valsalva, acquired aortic stenosis, mitral insufficiency, and aortic insufficiency were hospitalized here.

undertake the elective correction of vascular deformities in the juxta-cardiac area. As lessons were learned and experience and confidence acquired, techniques were developed which provided for the treatment of certain lesions within the heart itself. The great limitations imposed by the inability to visualize these areas combined with the obvious need for intracardiac visual techniques to broaden therapy to conditions then hopeless gave great impetus to the labors of those who hoped to correct deformities within the heart under direct vision. Hypothermia and cardiopulmonary bypass procedures made open cardiomy both possible and feasible and thus have permitted the correction or even cure of many previously hopeless lesions. In fact, in the space of but a few years, these advances have significantly altered the prognosis of virtually every serious cardiac condition, congenital or acquired in origin.

And what of the future? In this day of great technical advances, when science fiction is scarcely ahead of scientific achievement, can one see the limits of cardiac surgery? This entire field is moving forward so rapidly that the rash individual who says "It can't be done" is generally interrupted by someone else "doing it." In

medicine, there are no ultimate solutions to problems—only ways of doing things better—science is an endless frontier and thus we predict a continuing "performance beyond prophecy."

We are grateful to Doctor Clowes for having allowed us to read his paper while it was still in manuscript form.

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CENTRAL NERVOUS system damage following cardiac arrest may be lessened by hypothermia. As soon as the patient is brought to the recovery room, treatment is begun with the use of a blanket containing a circulating coolant. When necessary, cooling may be aided by subcutaneous injection of small doses of meperidine, promethazine, or chlorpromazine in amounts which control shivering but do not suppress respiratory or cardiovascular function. Therapy does not otherwise differ from customary postoperative care.

The interval between arrest and cooling should be as short as possible, although it is difficult to regulate. In survivors, the period ranges from one to three hours, and in nonsurvivors it varies between one and six hours. The depth of hypothermia is between 30 and 32° C. Although a greater amount of cooling may benefit the central nervous system, ventricular fibrillation is much more likely to occur when temperatures below 30° C. are used. Observation of the patient should determine the length of the cooling period, with therapy being gradually discontinued upon improvement.

In 19 patients with hearts in asystole or fibrillating, cardiac arrest occurred and cerebral injury was noted. Of the 7 patients not given hypothermia, only 1, or 14 per cent, survived. Of the 12 receiving hypothermia, 6, or 50 per cent survived. In those who survived, duration of hypothermia was thirty-four to forty-eight hours, while, in those who died, this period ranged from three hours to eight days. None of the survivors had residual neurologic deficits, and cortical blindness in 1 was completely cleared within six months.

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Some Aspects of the Problem of Farm Accidents

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THE NUMBER OF ACCIDENTS that occur on farms each year and the severity of the injuries which result have appalled physicians in agricultural communities for many years. I grew up in a rural section of Illinois, where my father was a physician in a large farming community. Among my earliest recollections are hurried trips with him in his horse and buggy to some farm where the farmer had been kicked by a horse, fallen from a windmill, or been overcome by heat during the haying season. As a high school and college student, I worked on our farm near town and experienced a few minor farm accidents myself. Thus, my interest in them is not only an academic one; I have been associated with the problem of farm accidents since boyhood.

Too many people have looked upon farming as a quiet, safe occupation, far removed from the hazards of factory and shop. However, "the cool sequester'd vale of life," of which Thomas Gray wrote in his poem of rural life, has become one associated with danger. According to the National Safety Council, of the 13,300 workers killed on the job in all industries in 1958 in the United States, approximately 3,300 lost their lives in farm work. Farm accidents caused more deaths than any other major industry. In the death rates per 100,000 workers in major industries, farming was exceeded only by construction and extractive industries. Agriculture, the oldest of occupations, continues to be one of the most hazardous.

Accidents associated with farm machinery caused the greatest number of deaths on the farmland and around service buildings, according to data furnished by the National Office of Vital Statistics for the years 1954 to 1956. These were analyzed by the Department of Agriculture.¹ A study showed that the 4 leading causes of the 155 farm accident fatalities in Minnesota during 1958 were (1) machinery, (2) falls, (3)

fires and explosions, and (4) firearms. Other causes are listed in the accompanying table.²

Agricultural changes during recent years have brought about new working conditions. Increased mechanization has extended the farmer's capacity to plow, sow grain, harvest crops, and take care of livestock, but injuries have always occurred on farms. We sometimes forget, in our desire to attribute farm accidents to machines, that our grandparents experienced and often died from injuries due to handling farm horses. Doyle,³ as recently as 1956, conducted a limited survey in 1 county during a period of six months, which revealed that 8 out of 29 accidents were associated with horses. Nevertheless, although horses are yet to be found on farms, even though in fewer numbers than formerly, the shift to machines has been an important factor in the increase in farm accidents. That there has been this shift is evidenced in statistics from the United States Department of Agriculture, which showed that, in the period between 1941 and 1952, the number of mechanical cornpickers in this country increased from 120,000 to 635,000, the number of grain combines increased from 225,000 to 940,000, and the number of tractors increased from 1,700,000 to 4,400,000.³ Nor do we need to consult statistic reports to be aware of this change. In many localities in our mid-western states, the shocking and stacking of grain are agricultural procedures which today's farm boys never experience.

REASONS FOR FARM ACCIDENTS

Why is the farmer especially liable to incur injuries from machinery and from accidents in general? Why does he experience a greater number of injuries than persons in his community who work in factories? The answers to such questions not only involve the problem of the mechanical features of modern agriculture but also a consideration of the farmer's highly individualistic occupation, which has aspects all its own. Several studies have considered facets of this problem. Young and Ghormley⁴ reported on

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TABLE 1
FARM ACCIDENT FATALITIES IN MINNESOTA IN 1958

<i>Causes</i>	<i>No.</i>	<i>No. occurring in course of farming operations</i>	<i>No. occurring in farm home and vicinity</i>
Machinery	39	25	14
Falls	31	5	26
Fire and explosions	25	5	20
Firearms	16	1	15
Drowning	5	—	5
Suffocations:			
From foods	2	—	2
From mechanical	11	—	11
Blow from falling object	9	5	4
Poisoning	3	1	2
Electric current	1	—	1
Nonvenomous animals	2	—	2
Cutting or piercing instruments	1	1	—
Farm vehicle	1	—	1
Hot substance (burns)	2	—	2
Other and unspecified accidents	3	1	2
Excessive heat	3	—	3
Lightning	1	—	1
Total	155	44	111

Source: Minnesota Department of Health

575 farm accidents, which, during a period of nine years, brought the injured person to the Mayo Clinic. Powers^{5,6} and Calandruccio and Powers⁷ discussed accidents sustained in a rural section of New York where there is much dairy farming. Creevey⁸ surveyed 370 farm accidents in an area near Cambridge, New York. Snyder^{9,10} discussed trauma in rural areas. Some of these authors' answers to the important question, "Why do farm accidents occur?" are included in the reasons suggested below.

Weather. The farmer works in all kinds of weather, whereas the factory worker performs his assigned duties in a building where ventilation and heating facilities provide a safer working environment. In order to withstand the rigors of the cold, the farmer wears extra clothing, which impedes his movements. Often these clothes are worn loosely. A farm lad wearing a loose sweater which belonged to his father caught one of its sleeves in a machine and lost his hand. Another farmer lost his life when, on an extremely cold day, the extra muffler he was wearing was caught in a machine. He was working by himself. When he didn't come to the house for dinner, his wife went to look for him and found that he had suffocated. If someone had been working with him, his death might have been prevented.

Lack of help. The fact that the farmer often works alone is surely an important factor. "Two hands are better than one" may be a trite expression, but in the matter of safety it is true. I recall a farmer who tried to lift a machine and injured his back; with assistance, the task could have been easily accomplished. The farmer who is fixing a machine may keep the engine running because he doesn't want to take time to shut it on and off. If he had an assistant, the inherent dangers from such procedures could be avoided.

The farmer deals with unruly animals, very often by himself. In this day of mechanical devices, he dispenses with the aid formerly given by a hired hand and is able to accomplish his work alone. Yet, by handling livestock alone, he endangers himself more than when he had assistance.

Machines. The farmer handles powerful machinery over rough ground. Driving his tractor at high speed over a pasture to reach another field, an acquaintance of mine struck the stumps of two trees hidden in the late summer's grass. His tractor was overturned, and he suffered severe injuries. In factories, many powerful machines can be bolted to the floor, and thus the danger of such a machine tipping over is obviated.

Seasonal tasks. The farmer's tasks vary with the season of the year. He no sooner becomes accustomed to one piece of machinery than the type of work which requires its use is completed, and he must familiarize himself with another. Thus, facility which results from repetition is lost. This is in marked contrast to the factory worker, who comes to know his machine thoroughly through continual use.

Lack of supervision. The farmer has no foreman supervising his work in most instances. The role of the foreman in safety programs in factories is well recognized. Recently, in a visit to a factory, I observed a foreman reminding one of his men to wear safety glasses while working with abrasives. The farmer has no one to remind him. I recall very well a farmer who lost an eye from an injury sustained while repairing his tractor. He later told me that he had a pair of safety goggles but hadn't wanted to take the time to go to the house to get them. Had there been an alert foreman present, the accident probably would have been prevented. The farmer may remove safety devices from his machines and then neglect to replace them. In most instances, this negligence would be noticed in a factory by fellow workers or the foreman.

Unsafe equipment. The farmer often uses old materials as a part of his thriftiness. Old boards

have too frequently been used to make a scaffold; a ladder has been repaired by nailing a piece of old lumber to replace a broken rung. Some persons who would never think of walking under a ladder because of an old superstition will, nevertheless, climb a ladder repaired with unsafe materials. This is one of many instances in which persons are "penny-wise and pound-foolish."

Overwork. Fatigue is a major factor in farm accidents. The farmer works on his own time and without interruption. Many times in the summer, when the evenings are long, he works far into the night. He may even depend on illumination from lights on his tractor or moonlight. He has no eight-hour day with scheduled breaks. Top¹¹ has called attention to the fact that the farmer, when working with horses, saw to it that they had periods of rest and thus received a period of rest himself. These rest periods do not occur as frequently in this mechanized age. In the community where I grew up, it was the custom for the farmer's wife to bring a lunch to the workers in the fields. Today, this custom has almost disappeared in many areas. The farmer becomes tired but often fails to realize it. The relation of fatigue to the occurrence of accidents is direct.

Infections. The farmer runs a greater danger of infections from injuries incurred than do workers in factories. The nature of the working environment heightens this danger; the farmer's hands are contaminated with soil. The prevalence of the tetanus hazard on farms is generally recognized, but the presence of other organisms must also be borne in mind. When manure is used as fertilizer, the soil can be expected to be overly contaminated, and the farmer's machinery and tools also can be heavily contaminated. He may not stop work to receive care for minor wounds. A farmer who incurred a deep wound of the thigh while cleaning his barn paid no attention to it except to tie a cloth around it. His wife urged him to go to a physician, but he said he hadn't time. He died from tetanus. In a factory, medical care would have been arranged for him. Accidents which the farmer considers trivial are generally taken care of in factories, but such care is not readily accessible to the farmer.

Carelessness. Probably the largest and most significant factor which underlies farm accidents is carelessness. Many farm accidents occur as a direct result of simple neglect, which, without a doubt, plays an important role in all farm mishaps. The farmer is often careless in repairing his machine. Even if it is in top working order

at the beginning of the season, makeshift repairs are often made in the field and proper parts are not always used. The farmer who fails to take the power take-off unit out of operation before dismounting from the tractor is an example of plain carelessness.

Untrained help. Very often when the farmer can obtain someone to help him, that person may not possess the necessary training in farm manipulations. Sometimes he may be required to use younger children who are not prepared to deal with emergency situations. It should also be mentioned that sometimes youngsters taken along by their fathers on a machine "for company" become accident victims. Tractors were not designed to carry extra passengers. In few occupations are children permitted to come close to operating machinery, and yet this occurs on farms. An editorial writer several years ago commented: "It is no wonder, then, that one-third of the 306 fatal tractor accidents reported by Wisconsin and Ohio to the National Safety Council over a period of several years involved persons under 20 years of age. One case in 10 involved a child under 5 years of age."¹²

Haste. Too often the farmer fails to recognize the importance of safety because he is racing against time. He may lose his hay crop if his work is delayed. Various studies have shown that the peak of agricultural fatalities corresponds to the busy harvest season of late summer and early autumn. Haste coupled with fatigue explains the frequency of accidents in late afternoon and early evening hours.

Variety of work. The farmer generally works with machines without special training and does a great variety of tasks. As Creevey⁸ has said, he "has to be a Jack-of-all-trades—planter, animal trainer, lumberjack, machinist, carpenter, plumber, builder"—and is called upon every day to perform a variety of duties. It is not possible for him to send for an expert. For many of the tasks he is ill-prepared, and he does not possess the proper tools.

SURVEYS OF RURAL ACCIDENTS

Two surveys made in counties of Minnesota in recent years serve as examples of a means to obtain interesting and valuable information regarding farm accidents. In order to direct an attack most successfully against farm accidents, it is necessary to obtain data and study them carefully. The two surveys briefly discussed here show the type of information furnished by such a procedure.

An accident survey in Jackson County, Minnesota, was made by the Jackson County Home

Council with the cooperation and direction of Audrey Vulcan, county home agent; Ray Palmby, county agricultural agent; and Glen Prickett, extension safety specialist. Completed interviews from 196 farm families and 131 town families were studied. The analysis showed that a quarter of the farm families compared to about an eighth of the nonfarm families reported accidents. With accidents occurring twice as frequently in farm families, there should be emphasis on farm home safety. Certain facts which came from this survey are summarized seriatim:

1. Accidents, defined as any injury requiring treatment by a physician and/or requiring a recovery period of one or more days, occurred in about one-sixth of the families during the twelve months previous to the interview.

2. The male members of the family stood the greater chance of having an accident, about one-third of which involved the husband and another third a son.

3. The bulk of the accidents occurred to persons between 15 and 30 years of age.

4. About one-third of the accidents reported occurred in November. They occurred more frequently at the beginning and at the end of the work week. Over three-fourths of the accidents occurred during afternoon and evening hours.

5. Driving and playing were the activities engaged in at the time of the accident in slightly more than one-fifth of the cases. Field work accounted for about one-seventh; housework, yardwork, and chores were responsible in less than one-tenth of the accidents.

6. Falling or slipping accounted for over one-third of the accidents reported. In slightly over one-fifth of the accidents, the victim was caught in or between or struck against an object; slightly less than one-fifth of those involved were hit by a flying or falling object.

7. In over two-fifths of the cases, the respondent to the inquiry indicated that hurry, carelessness, or a combination of the two was primarily responsible for the accident. Fatigue and faulty equipment were each mentioned in less than one-tenth of the cases. It is interesting to note that weather, so often mentioned as a major cause of accidents, was cited in less than one-twentieth of the cases.

A study and analysis of accidents reported by physicians in Renville County, Minnesota, to the Renville County Agricultural Extension Service office is interesting. It covers the period from April 1, 1955, through March 31, 1956. The physicians of Renville County cooperated wholeheartedly in the survey and submitted monthly reports on prepared forms. Renville County

residents were almost equally distributed as to place of residence, for 53.4 per cent lived on farms and 46.6 per cent were classed as non-farm residents, according to 1950 census figures. Certain facts noted in the report are listed herewith:

1. Rural accidents reached a spring peak in May, reached a top peak in July, declined in midsummer, and reached a third peak in October. Those familiar with farming operations will recognize these peaks as corresponding to 3 periods of farm work, that is, beginning of field work, haying and harvest, and corn-picking.

2. Almost 49 per cent of the rural accidents were associated with machinery, or 158 of the 324.

By no means is the concern regarding farm accidents only associated with mechanical devices. Drowning ranks high as a cause of fatal injury on farms. In fact, it accounted for one-seventh of the accidental deaths on farms during a two-year period recently. Most of the victims were children, with a large toll among those below 5 years of age. Wells, cisterns, troughs, dams, and the many farm streams as well as water-filled farm utensils are places where drownings occur on farms. The great number of guns owned by those in farm areas cause almost as many deaths annually as does drowning.¹³

SOLUTION TO THE PROBLEM

Even more important than the question of why farm accidents occur is the matter of a good solution to the problem. What can be done about farm accident prevention? What is being done?

During the first quarter of this century, there was an awakening of interest in and recognition of the hazards found in factory work, transportation, mining, and construction. Measures were adopted to promote safety. Legislation regarding safety and improved working conditions resulted from efforts made. The need for men trained in safety became apparent, and safety engineers came to fill an important place in industry's safety programs.

Because of its primarily individualistic character, there has not been the same degree of recognition or of supervision in the matter of agricultural accidents. However, when it became apparent that mechanization was contributing in such large measure to farm accidents, manufacturers of farm equipment joined forces with health and safety organizations in their activities to promote farm safety. The initial step in the process was one of education. This

procedure had a twofold aim: (1) to provide information regarding farm accidents, and (2) to create an awareness on the part of those engaged in agriculture concerning farm safety practices.

The first definite programs of farm safety education were instituted about twenty-six years ago. At that time, the Agricultural Extension Service announced farm safety activities through its 4-H clubs. Minnesota was the first state to participate in this safety program. Various rural organizations now carry on safety programs. I have spoken at meetings of the Grange, Farm Bureau, and men's clubs of rural churches on the subject of farm safety and know of the interest of such organizations in safety education. Many times the Parent-Teacher Associations in rural communities feature a "Farm Safety Night" during their year's activities. Farm publications contain a large number of articles on safety. Regional farm safety institutes have brought together representatives from local farm bureaus, 4-H clubs, and other rural organizations to discuss prevention of farm accidents related to a particular locality. Members of the Safety Council of the state, physicians, and representatives of the Agricultural Extension Service have assisted in meetings of this kind. As a result of these organized discussions, each area is able to plan its own farm safety program and discuss methods for its operation.

Since 1944, the last full week in July has been designated by the President of the United States as Farm Safety Week. Special emphasis is placed on the prevention of farm accidents throughout the nation. Although only one week bears this particular designation, the emphasis in this particular week is on safety throughout the year. Through intensive publicity and other educational media, residents of rural communities are presented with information regarding the accident hazards that lurk in their everyday surroundings. Suggestions for their correction or removal are made.

One of the most valuable and effective means of education in farm safety is the instruction of young people through 4-H clubs and high school courses in agriculture. Home economics classes also have participated in safety programs for girls who either now or in the future will live on farms. In classes of this kind, actual safety practices are learned and demonstrated. Farm safety projects which are undertaken in such classes give practice which is extremely worthwhile. In addition, they make the participants aware of the many ramifications of accident prevention.

The manufacturers of farm equipment are in-

terested not only in the design of safer machinery but also in the education of those who will use such equipment. Operation manuals emphasize safety measures; demonstrators and salesmen are instructed in the prevention of accidents.

Other methods of farm accident prevention have been advocated. Routine inspections of farm premises in an effort to detect potential hazards have been suggested. An investigation of the circumstances involved in each accident by the local health officer or someone from the Agricultural Extension Service has been discussed. Such methods have proved valuable in many industries. Just how effective they would prove in agriculture, because of the lack of controlled environment and adequate supervision, has to be kept in mind. Thus, farm leaders generally continue to stress education in farm safety as the important line of attack.

What is the role of the medical profession in this tremendous task? Anything preventable that has an effect on the health of people is a concern of the physician. We cannot immunize against accidents as we do against smallpox and other communicable diseases, but we can build up resistance to them through safety education. We can suggest the removal of causes of accidents if we learn what they are. For too long a time, persons have said of farm accidents: "I don't know why it happened. It just did." Accidents do not "just happen." Accidents that can happen, do. If a search is carefully made for the cause, it will be found in the greatest number of cases. It may be something in the environment, such as inadequate lighting, or poor materials, or it may involve the patient himself, such as a lack of knowledge or proneness to accidents.¹⁴

A rational approach to the problem is the use of epidemiologic methods which have proved valuable in solving problems of communicable disease. Gordon¹⁵ has described this approach to the solution of accident problems. The agent involved needs to be recognized, and the mechanism by which that agent comes into play must be determined. The combined effect originating from the host, the agent, and the environment has to be considered.

An example of the scientific approach to health problems of the farmer today is shown in the establishment of the Institute of Agricultural Medicine during 1955 at the State University of Iowa College of Medicine under the directorship of Dr. Franklin H. Top, head of the Department of Hygiene and Preventive Medicine. Staff members of the Institute are engaged in the study of significant problems affecting the farm worker. One of the studies this past year concerned corn

harvest accidents. The report presented information on the mechanical corn picker and its operation, and on 2 corn picker cleanout tools, 1 of which was invented as a safety device. It discussed field trials of the two cleanout tools and their results and commented in general on farmer behavior and the problems of corn harvest safety.¹⁶ This is one of many significant problems which can be undertaken by a group of research workers in cooperative studies.

Advice from a physician regarding safety is just as important a part of preventive medicine as a vaccination against a communicable disease. "Antibodies against accidents" take longer to develop, perhaps, but prove effective. Safety education programs in industry have proved their worth. The role of the physician as a valuable member of a community's team engaged in farm accident prevention has been well summarized:

"Most physicians are close to their patients and take a personal interest in their welfare. This relationship is even more pronounced in the rural areas, where the physician and the farm resident usually share more than a patient-physician affinity. Therefore, the rural physician is in an ideal position to proffer advice that may prevent farm accidents. Every rural physician should volunteer safety facts and fallacies. It may be too late when the information is sought."¹²

OTHER OCCUPATIONAL HAZARDS

Although the emphasis in this discussion has been on farm accidents, mention should be made of other occupational hazards on farms. Shryler,¹⁷ Berry,¹⁸ and Doyle³ are among those who have called attention to the danger of the newer insecticides and pesticides used on farms. The potential dangers of these toxic chemicals are always to be considered. Through ingestion, inhalation, or skin absorption, some of them may prove dangerous to farm workers. The hazards of their use are often not understood, especially if little attention is paid to the labels. In addition, the method of application may itself be dangerous. Industry has instituted proper control measures for the use of the many new chemical products, but the farmer working by himself might not take the time to institute such measures even if he could.

There is much current interest in the condition known as "silo-filler's disease." This occupational hazard, due to the inhalation of nitrogen dioxide in or near freshly filled silos, has been recognized in recent years among farmers.¹⁹

Sneid²⁰ is among those who have emphasized the many dermatoses which may occur among farmers. Whereas the industrial worker in the

factory is exposed to more or less specific irritants, the farmer has contact with a great variety of agents which may cause dermatosis. These include such things as plant and animal parasites; bites and stings of insects; excessive heat and moisture; sunlight; chemicals, such as fertilizers, of various kinds; and insecticides. Mosier,²¹ Top,¹¹ and others have listed the zoonoses as health problems which are peculiar to the farmer. The importance of measures of value in their control has been discussed by Meyer.²²

These are a few examples of the many health problems which are important to the health and safety of the agricultural worker and his family. Many of these problems need further exploration. Toxicology and proper application of chemicals used widely today in agriculture need to be kept in mind as well as the safety of the great number of mechanical devices. In all these problems, the importance of education in farm health and safety cannot be underestimated, nor can the role of the physician in farm health and safety education be minimized.

Copies of surveys discussed were provided through the courtesy of Professor Glen I. Prickett, extension specialist in farm safety, University of Minnesota.

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Pediatrics Since 1870

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PEDIATRICS as a separate medical specialty in the United States first gained definite recognition in New York City shortly after the middle of the nineteenth century. J. Lewis Smith was the first professor of pediatrics; he was appointed to that position in 1861 at the Bellevue Hospital Medical College. Abraham Jacobi is generally considered to have been the first great American pediatrician and was the dominant figure in American pediatrics for forty years. A native of Germany, he graduated from the University of Bonn in 1851. Due to political activities during his student days, he was forced to escape to England, coming to New York in 1853. He opened the first pediatric clinic at New York Medical College in 1860. In 1870, he was made professor of pediatrics at Columbia University College of Physicians and Surgeons. He wrote on almost all subjects relating to pediatrics. He was also credited with being the pioneer in the bedside teaching of pediatrics.

There were other notable pioneers in the pediatric field. Thomas M. Rotch became professor of pediatrics at Harvard University in 1888. L. Emmett Holt followed Jacobi at Columbia Medical School in 1902. He was also pediatrician-in-chief at the New York Babies Hospital, the first hospital in the United States caring only for infants and children. His *Textbook of Pediatrics* gained wide use and influence. It is of interest that the textbook is still being published by his son, L. Emmett Holt, Jr., himself an eminent pediatrician and professor of pediatrics, together with Rustin McIntosh, present professor of pediatrics at Columbia University.

John Howland founded the first full-time pediatric clinic in this country when he was appointed professor of pediatrics at Johns Hopkins Medical School in 1912, the year the Harriet Lane Home for Invalid Children was opened at that institution. Edwards A. Park succeeded Howland in 1927. He remained until his retirement in 1946. Howland and Park made a lasting

mark in pediatrics, both as investigators and teachers. Other famous pioneers in pediatrics were Kenneth D. Blackfan at Harvard; Grover F. Powers at Yale; W. McKim Marriott at Washington University, St. Louis; and Joseph Brenneemann at Chicago. During the early twentieth century, more medical schools added independent departments of pediatrics as realization increased that infants and children are not simply "little men" but have problems of development and diseases which differ considerably from those of adults.

In 1888, the Section of Pediatrics of the American Medical Association was founded. In the same year, the American Pediatric Society was organized with Abraham Jacobi as its first president. This organization is still active and includes within its membership the leaders in the pediatric academic field. The Society for Pediatric Research was originally organized as the Eastern Society for Pediatric Research in 1929. Its purpose is to foster pediatric education and research among academic pediatricians under the age of 45. Sectional and local pediatric societies organized during the earlier years of this century were the Central States Pediatric Society, the New England Pediatric Society, and the Northwestern Pediatric Society. The latter two are still active and flourishing. Many cities had their own local pediatric societies or sections. It was estimated that there were over 1,300 physicians in 1929 in the United States who limited their practice to infants and children.

The American Academy of Pediatrics came into being in June 1930 with an organizational meeting held in Detroit. The need for an independent and inclusive national pediatric society had been recognized for at least eight years prior to 1930, and numerous meetings and discussions had been held by leading pediatricians to that end. All specialists in the care of children were invited to be fellows of the Academy. Its original aim, to which it still adheres, was to devote the specialty of pediatrics to these main goals:

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(1) child health and welfare, (2) raise standards of pediatric education and practice, (3) encourage research in pediatrics, and (4) cooperate with all other child health groups for the greater good of children. The need for such an organization is attested to by the fact that the Academy has grown from several hundred members to over 5,000 fellows since its inception. It holds a national meeting each fall and a regional meeting each spring, both of which are well attended and of high caliber. The American Board of Pediatrics was established a year or two after the Academy in order to assess and standardize qualifications for specialized pediatric practice and for fellowship in the Academy.

The official journal of the Academy is *Pediatrics*, published by Charles C Thomas. It covers all aspects of clinical and research pediatrics and has an excellent section of special articles and editorials. Charles D. May is the editor. The *Journal of Pediatrics*, another excellent journal, is published by the C. V. Mosby Company. The *American Journal of Diseases of Children*, published by the American Medical Association, is another valuable publication. The *Archives of Pediatrics* is a long-established pediatric periodical.

REGIONAL HISTORY

Minnesota. In our own upper midwest, the University of Minnesota Medical School in Minneapolis and the Mayo Clinic in Rochester placed this area among the leaders in pediatric education and practice. Julius P. Sedgwick was the first professor of pediatrics at the University of Minnesota, formally organizing the department in 1915 and remaining there until his death in 1923. A native of Wisconsin, he obtained his medical degree from Rush Medical College, Chicago, in 1899. After studying abroad and then engaging in the practice of pediatrics in Minneapolis, he also served in France during the first world war with the American Red Cross. At the University of Minnesota, he carried on, among other things, an investigation of the relative merits of breast feeding and artificial feeding.

Clemens von Pirquet succeeded Dr. Sedgwick as professor at the University of Minnesota for a short time. Following his resignation, the position was filled by Dr. F. W. Schlutz, who remained as head of the department from 1924 to 1930.

Irvine McQuarrie became professor and head of the Department of Pediatrics at the University of Minnesota in 1930, where he remained until his retirement in 1954. He is a graduate of

Johns Hopkins Medical School. Before coming to the University of Minnesota, he had served as instructor of pediatrics at Yale, associate professor of pediatrics at the University of Rochester, and chief of pediatrics at Henry Ford Hospital, Detroit, Michigan. During his regime at Minnesota, the Department of Pediatrics became widely recognized as a teaching and investigative center. Probably more professors of pediatrics throughout the country had their training in McQuarrie's department than in any other department of pediatrics in the nation. A friendly and gracious person, Dr. McQuarrie has always had a wide range of medical interests. Among other subjects, he has made contributions on epilepsy in childhood and on water metabolism. Since his retirement, he has served in a consulting capacity at hospitals in the Hawaiian Islands and Oakland, California. John A. Anderson, himself a Minnesota graduate and former member of McQuarrie's staff, is the present professor and head of the department. He has also served in the same capacity at the University of Utah and at Stanford University.

Other distinguished names in pediatrics at the University of Minnesota and in Minneapolis and St. Paul are Walter R. Ramsey, Frederick C. Rodda, Edgar J. Huenekeens, Chester A. Stewart, Erling S. Platou, and Max Seham, to mention only a few.

Henry F. Helmholtz organized the Children's Department at the Mayo Clinic in 1920. He took his undergraduate work at the University of Wisconsin and obtained his M.D. at Johns Hopkins in 1906, after which he engaged in postgraduate study in Berlin and Vienna from 1907 to 1909. He was assistant professor of pediatrics at Rush Medical College from 1910 to 1920 and was medical director of the Infant Welfare Society during the same period. Like Dr. McQuarrie, Dr. Helmholtz was a gracious and friendly individual with a wide range of pediatric interests. He wrote extensively on pyelitis in childhood. Following his retirement as head of the Section of Pediatrics at the Mayo Clinic in 1949, he continued to work actively for the good of children as chairman of the Committee on Health of the National Association of Parents and Teachers and as a delegate to the World Health Organization. During a heated campaign in Fargo concerning the question of fluoridation of the city water supply, Dr. Helmholtz took the trouble to come to Fargo at this writer's invitation. He addressed an evening meeting and then appeared on a television program in behalf of fluoridation. The election took place with fluoridation winning by 300 votes

in a close contest. I have always felt that the impact made by Dr. Helmholz's visit and influence was enough to weigh the scales in favor of fluoridation of the water supply. Dr. Helmholz died in 1958.

Samuel Amberg was a friend and associate of Dr. Helmholz at Rush Medical College and came to the Mayo Clinic in 1921, the year after Dr. Helmholz arrived. Dr. Amberg was born in Germany in 1874 and took his medical education at the University of Heidelberg. He was associate professor of pediatrics at Johns Hopkins Medical School from 1910 to 1912. He was associate professor of experimental medicine at Rush Medical College from 1912 to 1921. Like Dr. Helmholz, he wrote extensively on many subjects dealing with childhood diseases. At present he lives in retirement at Rochester. He takes his place with other noted pioneers in the field of pediatrics.

In 1949 Roger L. J. Kennedy became head of the Pediatric Section of the Mayo Clinic. He has since retired from this post and has been replaced by James W. DuShane who, with a highly competent staff, is carrying on the best traditions of pediatric investigation and practice.

North Dakota. So far as is known, the first North Dakota physician confining his work entirely to care of infants and children was Ralph E. Pray of Fargo. He was a graduate of the University of Pennsylvania Medical School. He took a year's rotating internship in Philadelphia and then became associated with his father in the practice of general medicine and surgery in Valley City, North Dakota, in 1926. After a year, he decided to specialize in pediatrics and returned for an internship at the Philadelphia Children's Hospital and then went to Fargo to engage in group practice. He was the first North Dakota state chairman of the American Academy of Pediatrics. He remained in practice in Fargo until 1941, when he and his family moved to Salinas, California, where he later died. His brother, Laurence G. Pray, came to Fargo in the winter of 1941 and has remained there to the present. He is with the Fargo Clinic. His medical degree was obtained at Washington University, St. Louis, with postgraduate study at St. Louis City Hospital, St. Louis Isolation Hospital, St. Louis Children's Hospital, the Johns Hopkins Hospital, and the New York Babies Hospital.

Bernard A. Mazur entered practice in Fargo in 1937. A native of Buffalo, New York, he received his M.D. degree at the University of Buffalo. He took his postgraduate training at the Buffalo Children's Hospital. He is associated

with the Dakota Clinic. Ralph E. Dyson practiced pediatrics at the Northwest Clinic in Minot for a number of years before returning to Iowa, his native state, where he is at present practicing in Des Moines. Marlan H. Poindexter entered the practice of pediatrics in Fargo in January 1947. He is with the Fargo Clinic. His medical degree was obtained at the University of Kansas. His postgraduate work was at the Milwaukee Children's Hospital and at the Michigan Children's Hospital of Detroit. William B. Armstrong became associated with the Dakota Clinic in 1949. He has lived in Fargo most of his life. He obtained his M.D. degree from McGill University, Montreal, Quebec. His postgraduate pediatric work was done at the Children's Memorial Hospital, now the Montreal Children's Hospital.

Wayne E. Le Bien came to Fargo to engage in pediatric practice at the Fargo Clinic in 1949. He is a graduate of the University of Minnesota Medical School and took his postgraduate work at the University Hospitals and Minneapolis General Hospital. He is a native North Dakotan, his father having been a general practitioner at McHenry. Louis B. Silverman is in pediatric practice at the Grand Forks Clinic, Grand Forks, North Dakota. A native of Grand Forks, he obtained his M.D. degree at Rush Medical College. His postgraduate study was at the Children's Hospital of Michigan, Kingston Avenue Hospital for Communicable Diseases, Brooklyn, and the Beth El Hospital, Brooklyn. Robert B. Tudor is with the Quain and Ramstad Clinic in Bismarek. He is a graduate of the University of Minnesota Medical School and took postgraduate work in pediatrics at Johns Hopkins Hospital, Baltimore, and Duke University Hospital, Durham, North Carolina. Richard E. Dormont is with the Northwest Clinic, Minot. He graduated from Yale Medical School and took postgraduate work in pediatrics at Johns Hopkins Hospital and at the Charity Hospital, New Orleans. He came to Minot in 1953.

James V. Miles has established offices for pediatric practice in Jamestown. He took his specialty training at the Mayo Clinic. He is a native North Dakotan. Chris N. Christu has been associated with the Fargo Clinic for the past two years. A graduate of Louisiana State University Medical School, he took his postgraduate training at Johns Hopkins Hospital and at the Mayo Clinic. Samuel L. Pettit, a native of Pennsylvania, is with the Grand Forks Clinic. He is a graduate of Albany (New York) Medical School and took his postgraduate training there. Other pediatricians to enter practice in North Dakota

recently are William Kitto, Minot; W. G. Garrett, Bismarck; H. P. Smcenk, Bismarck; Hendrika A. Van Drunen, Bismarck; and John Sessums, Fargo. With one exception, all pediatricians in the state are in group practice.

CHANGING ASPECTS

Childhood disease and the practice of pediatrics have altered radically since Dr. Jacobi first established his pediatric clinic in New York City in 1860. At that time, Pasteur had not yet established the germ theory of disease with his work with fermentation, chicken cholera, anthrax, and rabies. Lister had yet to propose and confirm his theory of antiseptic and, later, aseptic for surgical wounds. The first appendectomy had yet to be performed. Koch was still to discover the tubercle bacillus. The diphtheria bacillus was first demonstrated microscopically by Klebs in 1883 and identified by Löffler in pure culture in 1884. Sanitation measures were lacking or incomplete. Pasteurization of milk was unknown. Deaths were commonplace from infantile diarrhea, diphtheria, scarlet fever, and other streptococcal diseases. Syphilis and tuberculosis were major problems. Infantile rickets and scurvy often occurred as a result of improper feeding, especially in the lower socio-economic levels. Pediatrics necessarily concerned itself with problems of disease and infant feeding. Many pediatricians now in practice can remember the presulfonamide and preantibiotic days when deaths from pneumonia, mastoiditis, septicemia, and meningitis were frequent occurrences. A physician treating children in that day needed to be familiar with the technic of tracheotomy and intubation for laryngeal diphtheria. There was little time or opportunity for pediatric specialists to consider growth and development or emotional and behavior problems to any great extent.

Following Pasteur's early work, the causes of many more diseases were found as research progressed in our medical schools and other scientific centers. By the time the sulfonamides were first put into use in the mid 1930's, the cause of most bacterial diseases and some viral diseases was known even though treatment for many of them was still asymptomatic. With the advent of sulfonamides, and later penicillin and other antibiotics, the tide turned in the battle against many serious childhood diseases. We no longer see the frequent deaths from pneumonia, meningitis, and related diseases. With better sanitation and better infant feeding, together with more effective treatment, feeding problems and diarrhea have settled down into

a relatively minor position. Widespread immunization procedures against whooping cough, diphtheria, tetanus, smallpox, poliomyelitis, and, sometimes, influenza have further lessened childhood morbidity and mortality.

All of this should not imply that diseases of infancy and childhood have been completely conquered. Emergence of resistant strains of staphylococci has caused serious outbreaks in newborn nurseries throughout the country. Resistant strains of other microorganisms have also emerged as successful treatment which has eliminated the traditional killers to a great extent. A number of new viruses have been identified as the causes of diseases which are threatening to take the place of the previously prevalent bacterial diseases. Pediatricians are still harassed by anxious mothers and feverish babies. In general, however, one may say that the situation is greatly improved over that of thirty or forty years ago. As an example, neonatal infant mortality has decreased during that time from 150 deaths per 1,000 live births to 20 deaths per 1,000 live births, with a similar improvement during later infancy and childhood.

As a result, it has been possible in recent years for pediatricians to turn their attention to other important problems of childhood. Accidents and poisoning are so frequent in children that poison control centers have now been established in almost all major communities in the nation. This has been largely brought about by the efforts of the American Academy of Pediatrics and its members, although other physicians, as well as the United States Public Health Service, have played an active role. More thought is being given to the proper training of pediatricians and their role as family advisers concerning mental and emotional problems as well as those of physical health and development. The new pediatrics which is being extensively written about and discussed should, therefore, include more than just regular physical examinations, laboratory tests, and immunizations. There are numerous opportunities for additional training and subspecialization in allergy, neurology, cardiology, hematology, diabetes, and psychiatry. The reward is a larger referral practice, with a greater number of interesting and challenging cases and proportionate financial return.

The new pediatrics can best be practiced in a group with other pediatricians and with other specialties represented for convenient consultation and laboratory and x-ray examinations as required. Association with other pediatricians makes it possible to allocate nights, week-ends, and vacations so that each member has an op-

portunity for sufficient relaxation and study and a chance to spend sufficient time with each patient to become acquainted with him personally and to learn something of his family relationships. The pediatrician, like the internist and general practitioner, is in a good position to serve as a substitute for the old-time family physician, who not only took care of medical problems but was a friend and confidant. Pediatricians, like all other physicians, must also undertake responsibility to the community as well as to patients if medicine is to continue to deserve respect. Donations to charities commensurate with income is only one facet of this re-

sponsibility. Pediatricians can make themselves available for talks to parent-teacher associations, child study groups, and others. They can participate in groups advancing the work of mental health and crippled children and cooperate with local and state health departments. Participation in the activities of local and state medical societies is a definite asset to those groups because of their specialized knowledge of child health. Physicians taking part in civic affairs reflect credit on themselves and on the entire medical profession. By adhering to all of these concepts, pediatrics can continue to play an increasingly valuable role in the family of medical specialties.

RHEUMATIC PNEUMONIA decreases the chance of survival in children with rheumatic fever, and those who do survive suffer from extensive residual heart disease. Prognosis may be improved by hormone therapy.

Symptoms of rheumatic pneumonia in children include anxiety, slight cyanosis, flared nostrils, grunting respirations, hacking cough, and fever. Respiratory rate may be as high as 100 per minute. The heart is enlarged, with distant heart sounds, rapid rate, and systolic and diastolic murmurs at the apex and right side of the base. On repeated chest films, pulmonary infiltration appears and disappears rapidly. Signs of myocarditis are usually found upon electrocardiographic examination. A hemoglobin value of less than 10 gm. per 100 cc. and a leukocyte count of more than 12,000 per cubic centimeter are common findings.

Postmortem examination of the lung may reveal fibrin in the alveoli and mononuclear infiltration. Fibroelastic proliferation may be seen in the alveolar fibrin.

Of 12 children given cortisone or prednisone, 5 survived. Of the survivors, all had mitral insufficiency, 4 had cardiomegaly, and 2 had aortic insufficiency. Larger doses of hormones had been given to the survivors than to those who died. Of 11 children not given hormones, 2 survived, both with cardiomegaly and aortic insufficiency.

D. GOLDRING, M. R. BEHRER, W. THOMAS, G. ELLIOT, and G. BROWN: Rheumatic pneumonia in children. *Postgrad. med.* 26:739-747, 1959.

Ninety Years of Cancer Progress

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... from it (history) one learns not only what has happened in the past, but also what may occur at the present, and what is likely to develop in the future. BUTLER, B.: *Cancer* 7:1, 1954.

“THE ABDOMEN, chest, and the brain will be forever shut from the intrusion of wise and humane surgeons.” This statement by Sir John Erichsen three years after publication in June 1870 of the first issue of *THE JOURNAL-LANCET*, then *The Northwestern Medical and Surgical Journal*, reflected the hopeless, defeatist attitude of the medical profession of that time in regard to the management of cancer.

In the preceding decades, fundamental advances in surgery and pathology by Billroth, Lister, Virchow, and others had prepared the way for more accurate and earlier diagnosis, for more effective treatment, and for the burst of progress in the management of cancer and in cancer research during the last three decades of the nineteenth and the first six decades of the twentieth centuries.

Ten years before the birth of *THE JOURNAL-LANCET*, Beale had published his study of cells exfoliated in the sputum of a patient with cancer of the pharynx, thus opening the science of exfoliative cytology, now so important in cancer diagnosis. Virchow and Bennett correctly described leukemia in the 1850's, and Hodgkin had published his description of “morbid appearances of the absorbent glands and spleen” in the preceding decade.

By 1870, then, the field was fertile for the cul-

tivation of the science and the art of the management of cancer. The status of cancer knowledge at that time was well summarized by Dunn of Minneapolis in an early issue of *Northwestern Lancet*, entitled, “A Brief (10,000 words) Review of the Cancer Problem.” He was of the opinion that there was little established knowledge concerning cancer beyond the fact that it leads to certain and disagreeable death. He was also certain that cancerous affections had increased in frequency during the past quarter of a century, and that they became fatal where floods prevailed and where vegetation was killed and decomposed. He knew that the influence of heredity had been much overrated, and that, in the relative or absolute cure of cancer, the earliest possible recognition was of the greatest importance. He was lukewarm concerning the value of pathology in determining malignancy. The microscope had not helped in the least in the prognosis of tumors of the pelvis, abdomen, and breast.

Concerning the then current forms of treatment—hard soap, condurango, chloride of potassa, Chian turpentine, creosote, acetic acid, caustics, ligature, congelation, pressure, and electricity—he stated that an enthusiastic advocate had reported that great improvement had occurred in many and cure in a few cases, while those not hypnotized by the faith failed to obtain any result. He recommended use of these modalities only in inoperable cases to fulfill the humane purpose of adding the buoyant influences of hope to a doomed life.

About the turn of the century, however, the sciences of chemistry, biology, physiology, pathology, and physics developed so rapidly in their applications to clinical medicine, including cancer, that there was a veritable explosion of discoveries benefiting the cancer patient. The rapid succession of discoveries by Roentgen, the Curies, Rutherford, and Becquerel laid the groundwork for our present atomic and nuclear

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developments, with their enormous value in cancer diagnosis, treatment, and research.

LITERATURE

To a great extent, progress in cancer is due to progress in communication. Advances made in one location are promptly disseminated through numerous journals, collective reviews, abstract services, and medical meetings—local, national, and international—devoted to cancer. In this country, cancer journals with the widest distribution are *Cancer Research*, *Journal of the National Cancer Institute*, *Cancer*, and *CA—Bulletin of Cancer Progress*, and the leading organizations sponsoring conferences and general meetings are the National Cancer Institute, the American Association for Cancer Research, and the American Cancer Society. Practically all other countries have organizations for the control of cancer and publish journals, for example, *British Journal of Cancer*, *Zeitschrift für Krebsforschung*, *Der Krebsarzt*, *Oncologia*, *Tumori*, *Neoplasma*, and *Gann*. The international organization's official journal is *Acta Unio Internationalis contra Cancrum*.

In addition to these special journals, articles on cancer appear in practically all medical journals and on many general medical programs. The lay press features special reports for public information. Therefore, cancer today is no longer concealed as a disgrace, and patients are diagnosed earlier when cure may be possible.

DEFINITION

As in many other fields of medicine, the terminology of neoplastic diseases is constantly changing. A century ago, the term "cancer" was even less specific than it is today. Any tumor that tended to progress and become fatal was a cancer, or a scirrhus. The discoverers of leukemia had no thought of classifying it with cancer. Today, the term "cancer" is applied to leukemia, lymphoma, carcinoma, sarcoma, and all other cellular tumors tending toward extension and fatal termination.

CHARLATANRY

The editorial of the first issue of *The Northwestern Medical and Surgical Journal* solicited letters "in any way correcting the tendency to charlatanry, which, to a certain extent prevails in this State, as in all the newer, and some of the older, States of the Union." Ninety years later we are still searching for ways in which to control quackery in cancer. Science and law are still ineffectual in preventing exploitation of the

cancer patient by either the sincere but misguided physician or the downright quack. In this country, elaborate mechanisms have been set up by the National Cancer Institute and the American Cancer Society for investigating the worth of new remedies; but, without legal compulsion, the promoters of a remedy need only refuse to submit it for study and continue to use it. However, since the last century, the public has gradually gained knowledge, making it less gullible concerning cancer "cures."

NATURAL HISTORY OF CANCER

"It is to be hoped that the pathogenesis of cancer will soon be removed, like that of tuberculosis, by the investigation of science, from the darkness of speculation into the broad daylight of exact demonstration." This hope, expressed by K. H. E. Cassel in the 1884 volume of *Northwestern Lancet*, is still unrealized today, although much progress has been made through the basic biologic, chemical, and physical sciences. It is generally acknowledged that the nature of cancer can be learned only through studies of normal cells and tissues and of the mechanisms of action of the many known carcinogens.

Much attention has been given to host resistance to malignant change. Detailed immunologic investigations in animals and man are being made. Differences in susceptibility have long been recognized.

Dunn's review, referred to previously, stated: "Two tumors identical in gross appearances and, indeed, in which the microscope can detect no essential differences may pursue the most divergent course. One may fail to affect the glands at the end of four or five years and produce no secondary growths for years, endangering life only at a very remote period, while another may in a few weeks or months involve the glands, rapidly become disseminated throughout distant organs, and speedily terminate existence." This variation in individual susceptibility and resistance to neoplastic change is being investigated experimentally in the laboratory and clinically. The basic differences in tumor growth are due to alterations in the host and not in the neoplastic cell. These little understood systemic factors, referred to in the early literature as temperament, vital tendencies, and internal climate—hormonal, neurotropic, immunologic, and metabolic—constitute the basis for the modern concept of biologic predeterminism. The latter term is a synoptic expression for the biologic balance, probably of genetic origin, between host and neoplasm established before the neoplastic process becomes clinically detectable. Full apprecia-

tion of this concept brings a note of optimism into the management of many chronologically late cases of cancer. Investigation of occasional, verified spontaneous regressions and cures is another possible source of knowledge of the natural history of cancer.

Search for fundamental differences between cancerous and normal cells and for the mechanisms developing these differences continues. To date no abnormal substance has been identified within the neoplastic cell; the differences are quantitative rather than qualitative. Such alterations of degree have been described in the metabolic enzyme systems, such as cytochrome and cytochrome oxidase, and in the nucleoproteins. Carcinogens probably act upon the genes to induce mutations. There can be little doubt that these cytochemical investigations are more likely to yield early favorable results than is any other field of cancer research.

ETIOLOGY

It is futile to fix the attention upon, or even to talk of, finding "the cause" of cancer, for cancer is merely a convenient term covering a multitude of abnormalities with widely diverse causes and manifestations. Despite this, there is now considerable knowledge concerning the etiologic factors of many forms of malignant neoplasms. The particular value of etiologic information is that it may be applied in the control of cancer through prophylaxis. Present knowledge of environmental, occupational, and other factors, if completely utilized, could appreciably reduce the death rate from cancer. There is reason to believe, for example, that lung cancer could be practically eliminated.

CARCINOGENESIS

Carcinogenesis is the process whereby a normal cell becomes malignant and produces a tumor. It is only in comparatively recent years that attention has been focused upon the actual cytochemical mechanisms of carcinogenesis. Knowledge of the fundamental processes concerned in the production of cancer has increased little in the past ninety years. We still do not know *how* soot caused cancer in the chimney sweep, although it has been identified as the etiologic carcinogen.

At the end of the last century, the carcinogenic action of ionizing radiations was demonstrated in the lesions of pioneering radiologists and their patients; we still have but meager understanding of the actual mechanism. More and more carcinogens are being identified among the new

chemical compounds of industry and of modern civilization, such as insecticides, weed killers, food additives, tobacco smoke, and air pollution.

DIAGNOSIS

Roentgenologic. Probably the greatest advance in cancer knowledge resulted from the discovery of the x-ray at the end of the last century. Before that, only cancers of the skin and easily accessible sites were recognized. Techniques were rapidly developed for diagnosing tumors of the gastrointestinal, genitourinary, and respiratory tracts and of the soft tissues and bones. With the invention of more powerful apparatuses and more accurate methods, the roentgenogram has come to be the most important diagnostic method for internal cancer, and recently it has been applied to detect the otherwise undiagnosable, incipient, and still curable cancer of the breast.

Histologic. Little was thought of the microscope as an aid in cancer diagnosis ninety years ago; now it is the court of last resort. All other methods of diagnosis are merely supplementary to, or confirmatory of, the histologic picture as shown in biopsy or in the operative or autopsy specimen. Without the frozen section technique for spot diagnosis at operation, the present cure rates would not be possible; without the present systems of histologic classification and grading of tumors, prognosis would be still less exact than it is.

Cytologic. Following the finding of abnormal cells in the sputum of a patient with cancer of the pharynx ten years before the first issue of THE JOURNAL-LANCET, this discovery lay dormant until revived half a century later, when it developed into the valuable science of exfoliative cytology in the detection of cancer. First applied to the detection of cervical cancer, this procedure has been rapidly extended to cancers of the stomach, lung, breast, and other sites.

Largely through the efforts of the American Cancer Society and the National Cancer Institute, physicians and technicians are being trained, and the application of the cytologic examination has been widely promoted in this country as a mass screening method for the detection of unsuspected cervical cancer. This program and the campaign for self-examination of the breast have contributed materially to the current reduction of death rates from cancer of those two sites. But, like all other methods of diagnosis, exfoliative cytology is only supplementary to histologic diagnosis by biopsy. Positive cytologic diagnosis must be confirmed histologically before therapy is planned. Recent use of fluorescent microscopy, with acridine

orange as fluorochrome, has greatly facilitated the technic of cytologic diagnosis.

Colposcopic. Visualization of the uterine cervix by means of the colposcope with a magnification of 10 to 20 times is an additional diagnostic procedure developed in recent years. Quite recently, magnification of 210 times was made possible by development of the colpomicroscope, by which the stained and unstained surface may be visualized through a shield pressed against the cervix to maintain focus. The advantage of this method over conventional cytology is that the cells are seen in situ in their living relationships. Here again, the method must be considered only as a supplement to confirmatory histologic diagnosis. Colposcopy is used less in this country and England than on the Continent.

Chemical. In cancer of the prostate, blood analysis frequently shows increase in serum acid phosphatase, especially when metastases are present. In cancer of the thyroid, diagnosis is facilitated by utilization of the affinity of thyroid tissue for radioactive iodine, and assays for iodine excretion may be indicative of the production of thyroid hormone. In interstitial cell tumors of the testis, 17-ketosteroid estimations are of diagnostic significance as are estrogen determinations in certain ovarian tumors and steroid assays in androgen-, corticoid-, and aldosterone-producing tumors of the adrenals. All of these tumor-hormone relationships have been found in this century. In the diagnosis of cancer of the stomach, determination of gastric acidity, preferably by use of the tubeless resin-dye excretion method, is often helpful. Chemical staining of glycogen on the surface of the uterine cervix is of value in diagnosis of malignant lesions. The applications of chemical methods to cancer diagnosis and research are numerous.

TREATMENT

Surgery. The beginning of serious modern surgery for cancer came about the time that THE JOURNAL-LANCET was first published. Standardized operative procedures were developed for each body site and closely followed until quite recently. The surgical objective, total removal of cancerous tissue, has been pursued through increasing numbers of extensive procedures made possible by discovery of more effective anesthetics, antibiotics, and acid-base balancing and by greater use of plasma and blood transfusions. In-block excisions are being made wider and wider to include more and more lymph chains. Such operations as pelvic exenteration,

hemipelvectomy, and "multiple-look" laparotomy were not possible before the basic improvements in operative conditions. Now even elderly patients, formerly deemed inoperable, are treated surgically and successfully for cancer. It is said that surgery has reached so great a degree of perfection that we must look elsewhere for improved survival rates.

Radiology. Shortly after discovery of the x-ray, it was put to use in cancer therapy, first in lesions of the skin, then of the breast, and later of the internal organs. With progress in x-ray machinery, increasing dosages were used, until today the supervoltages of the Van de Graaff generator, the radioactive cobalt unit, the betatron, and similar machines illustrate how far the physicist is ahead of the clinician. Many radiologists still prefer conventional medium dosage x-ray and radium for all cancer radiotherapy.

Modern developments in nuclear science have made available a whole series of radioactive elements useful in one or another form of cancer—phosphorus, iodine, cobalt, caesium, iridium, gold, strontium, sodium, yttrium, europium, and tantalum. Interesting applications of nuclear science are the treatment of glioblastoma multiforme by injection of nonradioactive boron, which has a selective uptake by brain tumor tissue, followed by activation in a thermal neutron beam and radiation hypophysectomy by injecting of radioactive yttrium as a last resort in the hormone therapy of inoperable cancer of the breast and prostate. In 1870, such ultramodern procedures would have been classed with a trip to the moon.

Endocrinology. Little was known concerning the endocrine system in 1870. There was some clinical knowledge concerning the thyroid and there had been some experimentation with inactive ovarian and testicular extracts, but the existing structure of endocrinology developed in the present generation and was applied to cancer therapy in the last decade. Treatment of inoperable prostatic cancer by castration and administration of estrogens was the first application. Soon there followed the treatment of inoperable cancer of the breast by oophorectomy, administration of androgens or estrogens, adrenalectomy, and, finally, hypophysectomy. Cortical and adrenocorticotrophic hormones have been found useful in certain leukemias and lymphomas. Thyroid and thyrotropic hormones used in conjunction with estrogens and androgens are said to increase their potencies. As yet, however, endocrine procedures are palliative, not curative.

Chemotherapy. It is to chemotherapy that

many workers in cancer look for the greatest new advances. Huge sums have been appropriated for developing this field by the Congress of the United States through the National Cancer Institute and by the American Cancer Society. Thousands of chemical compounds are being screened by many research laboratories for anticancer activity in animals. The few such substances found are then screened for toxicity in animals and, if safe, for anticancer potency in man. During the past decade, many substances found useful places in the treatment of malignant disease, particularly the leukemias and lymphomas. Among these are nitrogen mustard, triethylene melamine, triethylene phosphoramidate, triethylene thiophosphoramidate, urethane, Aminopterin, Amethopterin, 6-mercaptopurine, myleran, and the several androgens, estrogens, corticoids, and adrenocorticotrophic hormones. All of these were unknown to the readers of the early issues of THE JOURNAL-LANCET.

It is with thoughts in mind of possible "jackpot" products among those now under investigation that some more optimistic cancer research administrators from time to time venture pronouncement of a "breakthrough just around the corner." Since such a "breakthrough" is highly improbable through surgery or radiation, which have nearly reached their probable maximum effectiveness, it must come through chemotherapy, which has already demonstrated its value in prolonging comfortable life, if it is to come at all. The step from palliation to cure may not be so great. However, as has been demonstrated in the field of space science, huge appropriations alone do not ensure success.

Palliation. Great progress is being made in the care of the incurable cancer patient. Comparatively comfortable months and years are being added to the lives of formerly short-lived leukemic patients and others with rapidly progressing malignant disease. Surgical and radiotherapeutic procedures not designed to cure and all chemotherapeutic and endocrine procedures are only palliative.

Modern society is giving increased attention to the home care of the incurable cancer patient. Public health agencies and the American Cancer Society have developed programs of service that include palliative measures. Physicians are coming to accept their responsibilities toward the incurable and terminal cancer patient. The psychiatrist is very recently entering the field, especially in regard to the needs of patient and family at the time the diagnosis of cancer is made and when the patient is dying. Medical opinion is still divided, though unequal-

ly, concerning the duty of prolonging life in the face of agonizing imminent death. Before undertaking a palliative procedure in a hopeless, dying patient, the surgeon, radiologist, or family physician has come to assess its value in terms of comfort rather than longevity. Although little or nothing is gained toward the long-term control of cancer from the effort and expense put into palliation, civilized society accepts responsibility for such care of terminal patients.

Psychology and Psychiatry. Application of psychologic or psychiatric procedures in treatment of the cancer patient was practically unheard of before the present century. Now much attention is given to the emotional status of the cancer patient and his family. Psychiatrists are attempting to analyze the psychologic needs of the patient, the family, and the attending physician. Pros and cons are argued concerning the advisability of disclosing the diagnosis and prognosis to the patient, and much has been written concerning the psychiatric management of the dying cancer patient. All may be condensed into the golden rule.

Complete treatment of the patient considers the mind as well as the body. It has been said that a psychiatrist should be added to every cancer team of surgeon, radiologist, family physician, and pathologist, and that he and the clergyman are especially needed at the deathbed.

During the period of rehabilitation, the psychiatrist can assist in helping the cancer patient mutilated by surgery or radiology to adjust. Successful group psychotherapy among cancer patients has been reported. Proper psychiatric care may prevent suicide.

PROGNOSIS

In 1870, the prognosis of cancer was uniformly grave. A diagnosis of cancer was a death warrant. Today prognosis is much more hopeful. With increased communication and more precise classification and grading of tumors, the physician is able to make a more accurate appraisal of the probable outcome in a given case of cancer. Cure rates are increasing. Twenty years ago, 1 out of 7 treated cancer patients lived five years or more. Ten years ago it was 1 out of 4. Today it is 1 out of 3, and if all present knowledge of cancer were fully utilized by the physician and the public, the ratio could probably be 1 to 2.

REHABILITATION

Ninety years ago, rehabilitation as a surgical specialty was unknown. Peg legs, glass eyes, ear trumpets, and false teeth were the forerun-

ners of today's intricately devised artificial limbs for use after hemipelvectomy, of the elaborate oral and facial prostheses for the surgically mutilated cancer patient, and of the organized programs of psychologic rehabilitation of the laryngectomee and of the colostomy patient.

More and more physicians are recognizing that their responsibilities to the cancer patient go beyond diagnosis and definitive treatment to include the phase of convalescence and rehabilitation. Much has been done recently toward rehabilitating the laryngectomee by organizations of the laryngectomees themselves for the purpose of teaching esophageal speech and of learning to participate in social activities. Similarly, patients with colostomies have organized into groups for the purpose of softening the emotional trauma in other victims of cancer of the colon who require colostomies. Prosthetic substitutes properly adjusted can reduce to a marked extent the psychologic effect of mastectomy.

The surgeon, especially when operating for cancer of the mouth and face, looks ahead to rehabilitation of the patient. He consults with the prosthesis specialist before operating. Such niceties of surgical procedure were unknown even a very few years ago.

RESEARCH

Animal. Just a hundred years before the first issues of THE JOURNAL-LANCET, the first attempt was made to determine the nature of cancer by animal experiment. Juice expressed from a human breast cancer was injected into a dog, resulting only in infection. Twenty years before the birth of THE JOURNAL-LANCET, the first malignant tumor was transplanted from one animal to another. But definitive cancer research in animals began early in the present century, with the transmission of chicken sarcoma by cell-free filtrates.

Today these early beginnings have been developed into innumerable methods of using animals to obtain the answers to questions concerning the nature of malignant tumors and their treatment. In the recently organized cooperative study of chemotherapeutic compound screening, thousands of animals are being used. It is also in animals that carcinogenicity of compounds is determined. All studies of cancer transmissibility, of heredity, of cytochemistry, and of immunity are made initially in animals.

Immunology. Knowledge of immunology, especially as applied to cancer, is of comparatively recent origin. In earlier days of cancer research, when cancer was thought by some to be of bacterial origin, immunologic studies were made

of tumor-inducing organisms. Now that viruses are in the cancer-etiology ascendancy, they are being similarly studied.

Natural immunity and resistance to malignant tumors are being investigated. Recognition of wide variations in susceptibility to cancer has led to a wide search for an explanation which might lead to a method of cure of many forms of cancer.

Virology. The young science of virology was initiated by investigators active in this field today. Less than a quarter-century ago, important experimental results were reported from the laboratories of the University of Minnesota. Among these was the discovery of a viral agent in the milk of mice which plays an essential role in the production of breast cancer in these animals. Many pioneers were of the opinion that viruses may be etiologic agents for most, if not all, cancers, including cancer in man. Definite proof is now available that some animal tumors are caused by viruses, and there is no proof that viruses do not cause human cancer. Recent evidence of the infectivity of nucleic acids has necessitated revision of older definitions of virus to include nucleic acids associated with the genetic apparatus of the cell. Newly developed optical and electronic tools may be expected soon to advance materially the knowledge of viruses in relation to cancer.

Physical. The recent burst of physical research has invaded many heretofore unrelated fields, including cancer. All the advances and discoveries in radiation physics have been utilized in cancer work. Nuclear and electronic sciences have yielded instruments and materials invaluable in diagnosis and treatment and in basic science research as applied to cancer. Among the recent developments is the device for mechanical scanning of slides of exfoliated cells for cancer, markedly increasing the capacity of the cytology laboratory.

Research in physical science has developed the several supervoltage sources of radiation so useful in treatment of cancer.

Biochemical. Since the beginning of this century, biochemical methods have been employed in the search for some tangible chemical difference between the normal and the malignant cell upon which to base therapy. Inorganic and organic cell constituents have been assayed. Enzyme systems have been analyzed. Only quantitative variations have been found. Yet it is probable that considerable knowledge of importance concerning cancer will be obtained by such methods. It has been said that some cancer investigators have become so fundamental in their

approach that they appear to be looking for a cause of life itself and for a cure of the second law of thermodynamics.

Clinical. "This procedure should still be considered to be experimental" is an expression often used in clinical cancer work, although overt experimentation with human life is contrary to medical tradition. The greatest use of clinical research is determining the effectiveness of chemotherapeutic and hormonal compounds. Much surgical and radiologic research has been required to arrive at maximally effective procedures and dosages. Only with standardized classification and grading of tumors and with accurate and ample records from many investigators can benefit be obtained from clinical research.

Research organization. Today nearly every country has societies of physicians and laymen devoted to the control of cancer, and much of the advance in knowledge of cancer is obtained through the support of these organizations. Typical is the American Cancer Society, with its tripartite mission of research, education, and service. More than a quarter of its funds are expended on research. Its educational activities are directed to the medical profession and the public. It is largely through these activities that cancer information is disseminated in this country.

Meetings of specialists, clinical and research, are held periodically for developing a selected topic. Such a mechanism for dispersing cancer information has been developed in the present century. The National Cancer Institute has similar and much larger programs of research, recently extending even into foreign countries. In addition, there are in this country many cancer centers for laboratory and clinical research. The first to be organized was the Roswell Park Memorial Institute in Buffalo, which was soon followed by the Memorial Center in New York, the M. D. Anderson Hospital and Tumor Institute in Houston, the Institute for Cancer Research in Philadelphia, the McArdle Memorial Laboratory at the University of Wisconsin, the Division of Cancer Biology in the Department of Pathology at the University of Minnesota, and centers at many other universities.

ADVANCES IN CANCER CONTROL

The American College of Surgeons has done much toward the advance of cancer control by establishing a system of accreditation of hospitals and clinics for the diagnosis and treatment of cancer. Detailed minimum specifications for such things as personnel, equipment, and rec-

ords were prepared, and only hospitals and clinics meeting these requirements are approved by the College. Many institutions have greatly improved their management of cancer patients in their efforts to qualify.

Modern social acceptance of responsibility for the needy, the old, and the infirm has led to large-scale nursing and related services to the indigent cancer patient at public expense. These worthwhile humanitarian activities, however, contribute but little to the over-all solution of the cancer problem.

It is probable that no factor has contributed more to the advance of cancer control than modern methods of professional and public education. Provision has been made for improvement in cancer education in the medical schools and for informing the public of its responsibilities in early diagnosis, treatment, and prevention of cancer. Special aspects of cancer are discussed by authorities in study groups in order to disseminate information more quickly and to shorten the interval between research discoveries and their clinical applications in the control of cancer.

No real progress in cancer control was made until adequate records were collected and analyzed less than twenty years ago. Connecticut was the first state to obtain detailed records of all cancer patients. Other states have followed, and the federal government has made spot checks in 10 cities from which statistics for the entire country may be estimated. Efforts have been recently directed toward international standardization of records so that adequate numbers of all kinds of cancer cases may be collected from different parts of the world. Much can be learned from such statistics.

Study of geographic variations in incidence and natural history of cancers of various sites, together with knowledge of variations of customs, diets, and climates must eventually yield valuable cancer information. Such studies, organized internationally, are now proceeding.

Search for carcinogens in environment, occupations, and customs, has yielded enough information, if fully utilized, to reduce markedly the incidence and death rates of cancers of several sites. The lag between such discoveries and their utilization as preventive measures continues to be too great. Examples are food additives and preservatives, air pollution, and tobacco smoking.

As in every other disease, prevention is the ideal method of cancer control. Theoretically, it should be possible to prevent every cancer of which the cause is known and correctable, and

there are many. Most important now is lung cancer, which could probably be practically eliminated by control of air pollution and smoking.

Cancers of the skin, known to result from exposure to ultraviolet radiation, can be prevented by avoiding excessive exposure to solar rays. It has been stated that universal male circumcision and male and female coital hygiene would practically eliminate penile and cervical cancer.

Discussion of advances in any field of human endeavor in this wealthy country would be incomplete without a reference to increases in available monies. In 1944, the expenditure on cancer research in this country was about half a million dollars. By 1957, this figure had increased a hundredfold, and, in 1959, the figure had reached nearly \$100 million. Even this huge key has not yet unlocked the secret of cancer.

FUTURE TRENDS

In the American tradition, budgets for cancer research will no doubt continue to rise, and more and more expert minds will be applied to the

problem. Gradually, clinical application of the known facts concerning cancer prevention will be made. Knowledge of self-preservation from cancer will continue to spread among the population. Courses in oncology will become routine in the medical curriculum.

Laws will be passed for control of some of the known carcinogenic hazards in environment, occupations, and customs. Organizations for the control of cancer will continue to increase in size and in extent of influence. Additional chemotherapeutic compounds will be found to be effective in many types of cancer. But it is highly unlikely that a single *cure* for cancer will be found. Prevention and early diagnosis will continue to be of greatest importance in the control of cancer.

The history of cancer chronicled thus far is the history of errors, of illusions, of disappointments, of occasional triumphs. The smile of the Sphinx continues inscrutable. BETT, W. R.: Historical aspects of cancer, in RAVEN, R. W., editor: *Cancer*. London: Butterworth & Co., 1957, vol. 1, p. 5.

RADICAL EXCISION is preferred to irradiation with or without simple mastectomy in patients with primary breast cancer. Local recurrence is most common with central and inner quadrant lesions. Patients with primary tumors in these areas should probably receive mediastinal x-ray therapy after surgery.

When carcinoma is operable, internal mammary nodes are usually not invaded. Roentgen therapy may be given if the axillary nodes contain cancer cells. Unless ultraradical therapy is anticipated, biopsy of an internal mammary lymph node may spread disease and is of little value.

So far, there is no statistical evidence to indicate that simple mammectomy combined with radiation is superior treatment for carcinoma of the breast. Comparisons must be made between contemporary series, since earlier diagnosis and treatment will gradually improve the results of any type of therapy.

Microscopic examination of irradiated cancer of the breast or axillary nodes usually shows incarcerated viable cancer cells, which may spread.

Among 184 patients undergoing radical mastectomy, 1 died and 3 were not subsequently observed. The five-year survival rate was 66 per cent for the total group, 88 per cent when nodes were histologically negative, and 47 per cent in cases with positive nodes.

E. B. GRAY, JR., and T. J. ANGLE: Radical mastectomy for carcinoma of the breast. *New England J. Med.* 261:1310-1314, 1959:

The Law of Medical Practice Requirements in Minnesota

Some Historic Observations

RAYMOND SCALLEN

Minneapolis

LESS THAN one hundred years ago, Minnesota had no law which had been enacted by its own legislature governing the legal requirements for admission to medical practice. Yet, when Territorial Minnesota, not quite like full-panoplied Athena, sprang into being from the lands of Wisconsin and Iowa, glowing with the realization that Wisconsin had permitted a portion of the eastern boundary of the new territory to be the St. Croix rather than the Mississippi throughout this region, a partial suit of armor was provided, for the new territory possessed a ready-made Code of Laws—those of Wisconsin (sec. 12, Organic Act, 1849).

This Code of Laws contained, in chapter 19 on corporations, the earliest Minnesota law on admission to medical practice. It authorized the formation of county medical societies, declared them to be bodies corporate with power to examine students and to grant diplomas. No candidate for a diploma could be examined unless he was 21 years of age, had "at least a good English education," had "studied medicine at least three years with some respectable practitioner," and could "produce satisfactory evidence of good moral character" (sec. 6, chap. 19, revised statutes). There were no penalties for noncompliance and section 14 of the act provided: "This chapter shall not be so construed as to prevent any person from practicing physic and surgery within this Territory, who is not a member of said societies."

This was the Minnesota law for many years. It was not repealed or amended by the terri-

torial legislature, and it became a part of the laws of the State of Minnesota in 1858 under that portion of its new constitution called "Schedule;" nor did the new state legislate on this subject, even temporarily, until 1869.

In the light of the tremendous events that have taken place in the relatively short time of our state's history and the high place occupied by the medical profession in popular respect and scientific development, it is difficult to envisage the state of the profession one hundred years ago and to comprehend the conditions of those times or to perceive the difficulties confronting the pioneer practitioners in their efforts to protect the people of Minnesota from imposture by unqualified persons claiming skill in the healing art—persons who proclaimed their alleged thaumaturgy where all could read or hear but whose art was delusion and whose therapy was fraud. While this climate of confusion caused much concern in the minds of the pioneer physicians and surgeons, there had hardly been time for legislative activity on their part. There was a dearth of physicians, too, and the newness of the territorial status had hardly worn off before the new state became a part of the Union. Almost immediately, our state experienced the throes of an internecine war, with its very existence at stake. The souls and bodies of our people were sorely tried, and able doctors were needed at the front.

But, by 1869, the fair "Star of the North" state was giving promise of stirring expansion and development, and that year is of great significance in the history of medicine in Minnesota. It was in that year that the first meeting of the Minnesota State Medical Society took place in St. Paul on February 1 and 2, and the first piece of Minnesota legislation on the subject of medical practice requirements was enacted. As additional

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background to a consideration of the problems presented, it is interesting to note that in that year the society found that the numbers of "regular" and "irregular" practitioners in this state were as follows:

County	Regular	Irregular
Dakota	5	4
Dodge	4	2
Fillmore	11	8
Goodhue	8	4
Hennepin	18	21
Houston	4	5
Olmsted	8	6
Ramsey	24	21
Steele	5	4
Wabashaw*	8	4
Washington	4	5
Winona	20	9
Totals	119	93

* (Thus)

It is evident from discussions and medical comment of the time that quackery and charlatanism were rampant in the land and that measures had to be taken to stamp out these evils. They occupied high priority on the agenda of the meeting in 1869, which was opened by Dr. Thomas R. Potts as chairman and at which Dr. Samuel Willey of St. Paul was elected president. A resolution was presented to the meeting at the afternoon session on the first day and was offered by Dr. David Day. It read as follows:

Resolved by the State Medical Society assembled that in case the Legislature now assembled desire to protect the citizens of this State from quackery it is the duty of the Society to cooperate with the Legislature and lend its assistance in framing all needful laws upon the subject; that Drs. Willey, Sheardown and Stewart be appointed a committee as the organ of the Society for this purpose.

This resolution seems to have met with a favorable reception, and, shortly after the termination of the meeting, a bill for an act was offered to the legislature. It is significant that this first bill to be enacted into law in Minnesota was sponsored by Dr. Samuel Sheardown, state senator from Winona County, who had been elected treasurer of the Minnesota State Medical Society at its first meeting. In all likelihood, the act was somewhat hastily drawn, and the zeal with which it was brought forward may have exceeded the legislative draftsmanship. However, the proposed law was noble in purpose and chivalrous in title, for it was designated "An act to protect the people of Minnesota from empiricism and imposition in the practice of medicine and surgery." It became law, having been passed with

practically no opposition, and was signed by Governor Marshall, engrossed on the records of the secretary of state, and became the first law on this subject ever to be enacted in our state. Although it was approved in March, by its terms it was not to take effect until October of 1869.

Since it is not possible to reproduce a copy of an illustrative portion of the act in the handwriting of the original, we set out the complete text as it was printed in the Session Laws for 1869:

CHAPTER XLI.

March 9, 1869

An Act to protect the people of Minnesota from empiricism and imposition in the practice of medicine and surgery.

Be it enacted by the Legislature of the State of Minnesota:

SECTION 1. That it shall be unlawful for any person within the limits of said state, who has not attended at least two full courses of instruction, and graduated in some school of medicine either in the United States or of some foreign country, or who cannot produce a certificate of qualification from some state, district, or county medical society, and is not a person of good moral character, to practice medicine, in any of its departments, or perform any surgical operation for reward or compensation, or attempt to practice medicine or prescribe medicine or medicines, or perform any surgical operation for reward or compensation within the said state of Minnesota.

SEC. 2. Any person living in the state of Minnesota, or any person coming into said state, who shall practice medicine, or attempt to practice medicine in any of its departments, or perform, or attempt to perform, any surgical operation upon any person within the limits of said state, in violation of section one of this act, shall, upon conviction thereof, be fined not less than fifty dollars, nor more than one hundred dollars, for such offense, and upon conviction for a second violation of this act, shall, in addition to the above fine, be imprisoned in the county jail in the county in which such offense shall have been committed, for the term of thirty days; and in no case wherein this act shall have been violated, shall any person so violating, receive a compensation for services rendered. *Provided*, Nothing herein contained shall in any way be construed to apply to any person practicing dentistry exclusively.

SEC. 3. Any person who fails or neglects on or before the first day of October, one thousand eight hundred and sixty-nine, to file in the office of the clerk of the district court of the county in which he resides or keeps his office, a sworn copy of the certificate or diploma of some school or college of medicine, that he has attended at least two full courses, and graduated at such school, or a sworn copy of a certificate of qualification of some state, district or county medical society, shall be permitted in any court of this state to sue for or recover any compensation for his services, advice or attendance as a physician or surgeon, and the failure to file a sworn copy of such diploma or certificate, as above provided, shall be prima facie evidence that he has not attended or graduated at any school of medicine or received a certificate of qualification from any state, district or county medical society.

SEC. 4. Any person studying medicine with a preceptor, qualified as in this act above provided, shall have three years from the commencement of his term of study to comply with the provisions of this act.

SEC. 5. This act shall take effect and be in force from and after the first day of October, one thousand eight hundred and sixty-nine.

Yet, before the meeting of the next legislature and the next meeting of the medical society in the early part of 1870, something had happened. As it was later described, the law appeared to be a dead letter. With all the idealism of its purpose, it seemed like a man born out of time, for it was enacted before the people of the state had received sufficient education on the necessity for its enactment. In the history of this legislation, the next thing that challenges the interest of the observer is that it was repealed by the legislature in 1870 (Session Laws 1870, chap. 47) in very brief terms.

Was this repeal the result of a resurgence of militant *kwakzalvers* indignant at the attempt to curb their deceitful activities? Was it the result of a conniving cabal of purveyors of nostrums and of panacea profiteers? With the irony of juxtaposition, it appears in the Session Laws immediately after an act repealing the bounty on wolves!

The law of the jungle was not in the ascendancy nor were the emissaries of quackery in control. A logical explanation is found in the record of proceedings of the second annual meeting of the State Medical Society held in St. Paul on February 1 and 2, 1870.

In his presidential address, Dr. Willey, as a part of an extremely able summation of medical conditions in the State of Minnesota, told his listeners:

At the last session of the legislature an act was passed to protect the people from the evils of empiricism and quackery, and although I am informed by some of the members of that body that good has been accomplished in their respective neighborhoods by summarily arresting the career of imposters, yet the law was a defective one in many respects, with no provisions for its enforcement, and it has not met the expectations of the framers or of the people. . . .

It has been suggested that the law, which is practically a dead letter, should be properly amended and that as a part of the amendment, to make professional attainments and moral character of the applicant the only test for admission to practice without reference to any private opinions he may entertain.

President Willey indicated that these were simply suggestions, but they apparently reflected the judgment of the men of the society who had given sober thought to this matter and who now realized that there was much to be done in preparing the people of Minnesota so that they

could fully understand the evils attendant upon quackery and to constructively give calm and deliberate study to the preparation of a more comprehensive act regulating the practice of medicine in Minnesota so that, if it were tested, it would have a real chance of success. He also stressed this need for education, for he was confident that people who read the advertisements of these imposters would know them to be charlatans from the very claims that they made and that quackery could not survive once the people understood its evils.

In keeping with the spirit of the presidential address, a resolution was offered which is significant because of the inferences which can readily be drawn from it. It was introduced by Dr. J. C. Rhodes, and it was adopted by the society. It stated:

It is the opinion of the Society that laws so formed as to protect the people from impositions and unqualified pretenders to the practice of medicine and surgery would result in good, and further we believe it [such a law] could be so worded as to afford such protection; but we wholly disclaim any desire upon our part to have such laws on account of any benefit to us as medical practitioners.

A committee consisting of Drs. Charles N. Hewitt, A. E. Senkler, Charles Hill, and C. H. Boardman was appointed to study and report, and it is evident that the further deliberation of the meeting brought forth the futility and potential hazard of amending the law. This was its report:

Your committee after serious deliberation expressed the belief that laws regulating the practice of medicine do not and can not reach the cause of quackery among the people but that that cause is to be found in the deficient knowledge on the part of the people in regard to the real ground upon which the practice of medicine rests, and that so long as that deficiency exists, quackery will flourish. If legislative action is to be had, it should be directed to the removal of that cause.

The report was adopted, and it reflected the careful judgment of these sincere medical men.

It is a good general who can perceive the necessity for a retreat in good order and consolidate his forces from a new position. While there was no express resolution committing the society to specific action, it is not difficult to infer that, if indeed these good doctors did not initiate the repeal, they did not oppose it. Also, it should be kept in mind that at the time of the 1870 legislature, in the Senate was Dr. Charles Hill of Pine Island and in the House was Dr. Albert C. Wedge of Albert Lea. Dr. Wedge had served with the 3rd Minnesota regiment as a surgeon, was a representative at the 1870-1871 session, and a state senator in 1879 and 1881.

Dr. Sheardown was not a member of the Senate in 1870, but it is highly significant that the repeal was introduced by Mr. J. M. Cool, a farmer who lived in the neighborhood of Dr. Sheardown and who was a member of the House of Representatives from Winona County.

There is no record of arguments at committee hearings or of debates with reference to the merits of this bill, which finally passed the Senate after having previously been passed in the House and became effective February 24, 1870. It seems clear, however, that with such strong representation in the legislature, vigorous opposition could have been made to the repeal measure.

It seems reasonable to assume in these circumstances that these sound medical men, alert to the realization that the act of 1869 "did not achieve what had been hoped for it" and, on further deliberation, that an attempt to amend it might make matters worse, simply decided that the remedy of radical surgery was indicated and that postoperatively a régime of education would have beneficial effect on the body politic.

It is to the great credit of these pioneers in establishing medical legislation in Minnesota that they were so completely realistic and objective in refraining from multiplying confusion by adding to it the disastrous results of legislation which was not completely adequate for the subject nor sufficiently prepared for acceptance by the people of the state—factors which are essential to the success of any law. Yet, they had made a step in the right direction and a step which stimulated thought and inspired study and preparation for later enactments. This alertness is evidenced by the fact that, in the 1871 meeting of the society, after considerable discussion with reference to the need for a law on dissection, although some were in favor of it, many thought it premature and the question was not pressed.

As a result of the lessons that had been learned in the course of initiating this legislation, by the time the 1883 legislature met, suitable preparations had been made and a most interesting representation in the legislature gave rise to the Session Laws (chap. 125) of 1883, to which we will later refer.

While Wisconsin has been given some recognition with reference to the question of boundary lines, recognition should now be given to the State of New York, for it was from that state and from the Albany Medical College that three determined physicians had come to Minnesota and later were elected to the Minnesota legislature for 1883. The gentlemen making up this

medical triumvirate, who brought their knowledge of medical science to their tasks as solons, were Dr. J. M. Wheat of the class of 1853 of the Albany Medical School, who settled at Lenora in Fillmore County in 1856; Dr. Robert O. Craig of the class of 1855, who settled at Waseca in 1866; and Dr. William L. Hollister of the class of 1861, who started his practice at Austin in 1868. We regret that space does not permit printing the text of this historic act.

To summarize, however, the faculty of the Medical Department of the University of Minnesota was established as a Board of Examiners with power to administer oaths, to take testimony, to conduct examinations, and to issue certificates. Graduates in medicine could be certified on their diplomas, but nongraduates were to be given an elementary and practical examination. The board could refuse certificates to "individuals guilty of unprofessional or dishonorable conduct," and it could revoke certificates for like causes. In cases of refusal, an appeal was given "to the body appointing the board." Practicing medicine was defined as professing publicly to be a physician, prescribing for the sick, or appending the letters "M.D." to the person's name. Students in charge of preceptors and army and navy surgeons were excepted. Curiously, the act provided a license fee of \$100 a month for itinerant vendors of any "drug, nostrum, ointment or appliance" intended for treatment. Practicing medicine in violation of this law was punishable by fine or imprisonment, and passing off another's diploma was made a felony.

It was noted in the presidential address at the meeting of the society in 1884 that this law had been enacted and that the profession considered it a very fair law and was much gratified at its passage.

But would it stand the test of objections to its constitutionality? This test was not long in coming. The first decision of our Supreme Court that bears upon this matter of medical licensure and rules directly on the constitutionality of the law of 1883 is entitled *State ex. rel. David F. Powell vs. State Medical Examining Board*. It is reported in 32 Minn. 324, 20 N.W. 238. The relator Powell petitioned the District Court of Ramsey County to order, by the writ of mandamus, the State Medical Board to issue a license to him. The court quashed the writ, which had the effect of a dismissal, and the appeal followed. Although the relator presented a diploma, which the board found to be genuine, showing that he was a graduate of a medical college in Kentucky and that he had passed the prescribed

course of study, the board refused to license him on the ground that he was guilty of unprofessional and dishonorable conduct by advertising himself in the newspapers and claiming in such publications the proprietorship of certain specific remedies. He claimed one such remedy would cure cholera morbus when taken internally and rheumatism when applied externally, which claims the board determined were untrue and impossible.

The court addressed itself immediately to the constitutionality of the act and held that, while the vocation of physician is itself lawful, the legislature, in exercising its general police power, may subject that right to reasonable restrictions and that, even though this is a qualified right, it may not be arbitrarily or without reason denied. The court further said that, while the act did not prescribe the manner in which the investigation and hearing of the applicant should be conducted, it held, in essence, that there should be a reasonable opportunity for the party interested to be heard. Also, the fact that the act provided for the Board's taking testimony in all matters relating to its duties and that a right of appeal was given indicated a compliance with the constitutional requirement of due process of law. And the court said (page 327):

... the legislature has surely the same power to require, as a condition of the right to practice this profession, that the practitioner shall be possessed of the qualifications of honor and a good moral character, as it has to require that he shall be learned in the profession. It cannot be doubted that the legislature has authority, in the exercise of its general police power, to make such reasonable requirements as may be calculated to bar from admission to this profession dishonorable men, whose principles or practices are such as to render them unfit to be intrusted with the discharge of its duties. And as the duty of determining upon these qualifications, both as to learning and skill, and as to honor and moral fitness, must from necessity be committed to some person or body other than the legislature, we see no reason why it may not be committed to the legally-constituted body of men, learned in this profession, named in this act.

The court further said (page 328):

We are referred to no decision, and we have found none, sustaining the position of the relator, that an adverse determination of such a body upon such a question, by reason of which the applicant is precluded from engaging in the practice of his profession, deprives him of his property without due process of law, or that such enactments are for any reason unconstitutional. On the contrary, such enactments have been repeatedly enforced, and their constitutionality sustained, in cases involving a consideration of those provisions relating to the mode of determining the qualification of the practitioner in respect to learning.

The court reached the conclusion that the law

under which the board acted was constitutional and that it was apparent that the applicant could not compel the issuance of a certificate to him by a writ of mandamus. It held further that the board had decided that questions presented by the application and the correctness of that decision, involving the exercise of the judgment of the members, could not be brought into review by this proceeding and were not properly before it. The court referred to the appellate provisions contained in the act itself.

The basic principles of that decision and of the law which it construes have been upheld many times since and as late as 1956, when the Supreme Court of Minnesota cited with approval this landmark decision in *Reyburn vs. Minn. State Board of Optometry*, 247 Minn. 520, 78 N.W. (2d) 351, and decisively reaffirmed the doctrine of the *Powell* case as a part of the settled law in this state.

The law of 1883 stood unshaken as a sturdy bulwark against other assaults in 1885 when our Supreme Court in *State of Minn. ex. rel. Edward D. Chapman vs. State Board of Medical Examiners*, 34 Minn. 387, 26 N.W. 123, upheld the board in its revocation of a certificate. The decision was written by Mr. Justice William Mitchell, one of the great justices of our Supreme Court, and a study of the full text is highly recommended.

There was likewise the companion case, *State of Minn. ex. rel. E. C. Feller vs. State Board of Medical Examiners*, 34 Minn. 391, 26 N.W. 125, which upheld the board in its decision that a physician who publishes an advertisement containing false statements as to his ability to cure disease, knowing these statements are false and intending to deceive the public, is guilty of unprofessional and dishonorable conduct within the meaning of the law of 1883. We quote from that decision (page 390):

The argument of counsel for relator proceeds upon the assumption that all that is charged is a breach of a rule of professional ethics by simply publishing an advertisement of relator's business as a physician. But this is a mistake. The complaint sets out, in full, the advertisement in which relator, among other things, asserts to the public his ability to speedily cure *all chronic*, nervous, blood, and skin diseases of both sexes, also *all* diseases of the eye and ear, without injurious drugs or hindrance from business; *all* old, lingering constitutional diseases, where the blood is impure, causing ulcers, blotches, sore throat and mouth, pains in the head and bones, *cured for life*, etc. The complaint further charges that relator published this advertisement for the purpose of soliciting and procuring, wrongfully and fraudulently, patients to submit themselves to medical treatment by him; and that the statements therein contained are *false*, and that relator *well knew* them to be false when he made them, and that it was intended thereby to deceive

the public and impose on the credulous and ignorant. The gist of this charge is, not that he advertised his business, nor merely that the statements contained in the advertisement were false, but also that relator *knew them to be false*, and made them *with intent to deceive and impose on the public*. If true, this is unprofessional and dishonorable conduct of the grossest kind.

And in *Stewart vs. Raab*, 55 Minn. 20, 56 N.W. 256, which involved the same law, the court held that a certificate to practice medicine issued by the Board of Examiners under the law of 1883 authorized the holder to practice his profession in all its branches. The defendants, in a suit for collection of a surgeon's fees, asserted the ingenious defense that the certificate which authorized the doctor to pursue the practice of medicine in this state did not authorize him to practice surgery. On this point, Mr. Justice Collins, speaking for the court, wrote:

Laws 1883, ch. 125, is entitled "An act to regulate the practice of medicine in the state of Minnesota," and the first section prescribes that every person practicing medicine in any of its departments shall possess the qualifications required by the act. To persons possessing these qualifications, certificates shall be issued by a board of examiners, and these certificates authorize the possessors to practice "medicine and surgery" in this state. The terms "practice of medicine," in the title of the act, and "practicing medicine," in its first section, are used in the broad and popular sense in which they are generally understood, applied, and, in fact, defined. One practicing medicine practices "the art of preventing, curing, or alleviating diseases, and remedying as far as possible the results of violence and accident." Therapy is the treatment of disease, and surgery is therapy of a distinctly operative kind. The plaintiff's certificate, in terms, authorized him to pursue the practice of medicine under the conditions of the act of 1883, and necessarily this included surgery. There is nothing whatever in appellants' point that, because surgery was not expressly mentioned in the certificate, plaintiff violated the law when performing surgical operations. The statute in question does not require a license or certificate for each department of medicine.

By the time the 1887 session of the legislature met, a sentiment had apparently developed in the profession for some changes in the law and for certain specific provisions with reference to the examinations. A bill was introduced in the legislature which underwent some changes by amendment before a final act was agreed upon. In the first bill, there was a provision which seemed to represent some of the feeling of the day that there should be a separate examining board from that which was provided for in the earlier act, and among the provisions of the first bill was one stipulating that no person engaged in teaching medicine should be appointed to the new board which was envisaged under the act. This language was stricken out, and the amended bill contained the provision that a board

member should not be a member of a college or university having a medical department. The first bill to be introduced gave physicians whose practice extended into the territory of this state from an adjoining state or territory the right to practice under the law. It only required them to make application to the clerk of the county in the state in which the county seat was nearest to the residence of the applicant. This was stricken out when the bill reached the House of Representatives. A provision was also added to the bill in the House which prohibited this act from applying to dentists. In the earliest draft of this proposed measure, after providing for the appointment of a board by the governor, it contained the provision that at least two of the members of the board may be homeopathic physicians. This was changed on amendment to read "shall" instead of "may." Whether or not this change had any relationship to the fact that the Minnesota Homeopathic Medical College was founded in Minnesota in 1886, this provision became a part of the final act.

In this connection, when petition was made to the Board of Regents of the University in 1887 urging the establishment of a high grade teaching department of medicine in the University of Minnesota, J. Arthur Myers, M.D., writes in his able work, *Invited and Conquered* (pp. 77, 78), "in February of 1888, the faculties of the Minnesota Hospital College and the St. Paul Medical College appeared before the Board of Regents in support of the petition; they offered to surrender their charters and tendered their properties for the temporary use of the state. Soon thereafter, the Minnesota College of Homeopathic Medicine made a similar offer." The original bill also had a provision for dispensing with the examination when the diploma had been issued more than five years prior to the time of the examination, but this was stricken out, as was the provision "effective on passage," the date of July 1, 1887, being substituted. An inspection of the original bill indicates the informal method of penciling in amendments, and material was stricken out by drawing a line through the rejected words or by posting over amendments which superseded them. The many interlineations and changes on the face of this bill show that the proposed law was the subject of much study and debate. Finally, a bill was agreed on which became chapter 8 of the Laws of 1887. This law was to be the fundamental law of Minnesota for the next fifty years, for it stood without substantial change for that period. It contained the following provisions: (1) the governor was given appointive and reviewal power

of a board of 9 members; (2) board members could not be members of a college or university having a medical department; (3) two board members had to be homeopathic physicians; (4) a list of detailed examination subjects was required; (5) the consent of 7 of the 9 members was required before an applicant could succeed; and (6) elimination of admission by diploma.

This law was challenged in *State vs. Fleischer* (1889) 41 Minn. 69, 42 N.W. 696, particularly with reference to the provisions of the act requiring the consent of 7 of the 9 members. The appellant focused his attack on this law on constitutional grounds, for, since he was convicted of practicing medicine without a license, he claimed that the 7-member consent proviso of the law clothed the board with an arbitrary right to disregard the qualifications of the applicant. On this point the court said (p. 70):

From the spirit and object of the act, plainly seen in its several sections, it is obvious that the lawmakers intended to establish a high standard of qualification and fitness for the medical profession, whereby the people might be protected from ignorance and quackery. In creating the board, before which all persons desiring to practice medicine must appear for an investigation as to skill and ability, it was within the legislative discretion to require that all, or more or less than a majority, of its members should participate in the examination, and, before issuing a certificate, affirmatively pass upon the merits of each applicant. The requirement that at least seven of the nine members must concur and approve, is a slight innovation upon the majority rule found in subdivision 3, section 1, c. 4, Gen. St. 1878, which would prevail in the absence of another. It is a mere declaration of the mode in which a determination must be reached.

In 1895, the number of homeopathic physicians on the board was increased to 3, and it is interesting to note that this provision was retained in the law until 1927. There was also a proviso that, after 1899, it would be necessary for all persons seeking to practice medicine and surgery to submit to an examination in the branches which are set forth in detail in the act of 1887, and they were also required to present evidence of having attended four courses of lectures at a medical college recognized by the State Board of Examiners of at least twenty-six weeks each, with no two courses in the same year.

In 1905, the legislature passed the Reciprocity Law, and this was the subject of litigation in the case of *Williams vs. State Board of Medical Examiners* (1913), 120 Minn. 313, 139 N.W. 500. The applicant in that case, previously licensed in another state, appealed to the District Court of Ramsey County because of the action of the Board of Examiners denying him a license to practice medicine and surgery in Minnesota.

The question directly involved was whether an appeal was provided by law in the case of a physician who came from another state and whose application for licensing was denied. Before this case was decided, the court very thoroughly examined the history of the development of medical licensing statutes and held that the right of appeal was a creature of statute, that no appeal had been provided for in the law establishing the right of reciprocity, and that, if the legislature had so intended, it would have included a right of appeal in the law that it enacted. While the legislature in 1909 had amended the general law to provide for an appeal to the district court rather than to the governor as previously provided, the Supreme Court held that this provision did not apply to a person claiming a right of license under the Reciprocity Act. To cure this apparent omission in the law, the legislature in 1913 by chapter 139 established such a right of appeal.

In 1917, the statute prohibiting fee splitting was adopted, becoming chapter 365 of the laws of that year. Subsequently, other amendments were passed, usually of an administrative nature and not important to this discussion, and it was not until 1927 that any substantial change was effected.

That was the year of the passage of the Basic Science Law and is a year of great significance in the field of medical legislation. Since the scope of our article does not concern itself with the Basic Science Law because of its wide application and since in that year the legislature adopted as chapter 188 some very sweeping changes in the medical practice law, we are limiting our observations to the provisions of that act. While a presentation of the full text is essential to a complete study of this subject, to conserve space we mention only some of the most important provisions of the law.

Although the law of 1927 continued the provision for vesting power of appointment in the governor and reduced the number of board members from 9 to 7, leaving out the proviso requiring the presence of homeopathic physicians, it provided that appointments to the board should be made by the governor from a list of recommendations given him by the council of the Minnesota State Medical Association and that, when vacancies occurred, the governor should fill them from a list recommended by the council of the association. The law among other things defined "immoral, dishonorable or unprofessional conduct." It further provided that, under the Reciprocity Law, a person licensed by the National Board of Medical Examiners should

have comparative status with a doctor who had been licensed by the board of another state. Also, the act exempted from liability for unlawful practice those persons authorized to practice healing as long as they confined their activities within the scope of their respective licenses and stated that the prohibitive provisions of the law should not apply to persons who "endeavor to prevent or cure disease exclusively by mental or spiritual means or by prayer."

In 1937, by chapter 203, the provision was added to the foundational requirements that the applicant for examination should prove "that he is of good moral character and that he had completed four sessions of not less than thirty-six weeks each at a medical school recognized by the board with the results envisaged in the previous legislation." This law gave the board power to suspend, in addition to that of revocation, and gave a right to a licentiate to appeal to the district court of the proper county on questions of law and fact, the same as was given to a regular applicant for examination. It also made an additional specification of "immoral, dishonorable and unprofessional conduct" by adding "Conduct unbecoming a person licensed to practice medicine or detrimental to the best interests of the public."

In 1957, provisions were made for temporary certificates for graduate training, providing for permitted activities under the temporary certificate and limiting the time for which the certificate could be issued, and, at the discretion of the board, permitting renewals and providing also for suspension or revocation of certificates. With reference to resident physicians, a proviso was added stipulating that temporary certificates should not be required of a doctor of medicine duly enrolled and regularly attending the graduate school of Minnesota, including the Mayo Foundation.

In 1959, the board was empowered in reciprocity cases to exercise discretionary power, the mandatory "shall" being changed to "may," so that the board could license a physician from a state which was not truly reciprocal with regard to Minnesota physicians who sought licensure there.

SUMMARY

We have endeavored to present the beginnings and the development of the law of legal requirements for medical practice in Minnesota, and, with the benefit of the perspective of history, we discern how well those who pioneered in this field met the challenge of the times and set a pattern for the future—a pattern which has been devotedly followed. While there have been developments in the law so that it could keep pace with the progress of science and the growth of our state, the comprehensive code of today's statutes serves to heighten our perception that the essential principles of earlier legislation have not really changed but rather have been strengthened in order to attain and consolidate the objective of establishing and maintaining high standards in the medical profession for the general good of our community.

ACKNOWLEDGMENTS AND SOURCE MATERIAL

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Source material: statutes and session laws of Minnesota, including Minnesota statutes annotated; decisions of the Supreme Court of Minnesota; *Transactions of the Minnesota State Medical Society*; legislative manuals; newspapers of the period; "History of medicine in various Minnesota counties" commencing January 1938 in *Minnesota Medicine* by John M. Armstrong, M.D., and members of his committee; records of the secretary of state and of the state archivist, including original bills for acts; Minnesota biographies and historical articles; "Minnesota Medical Legislation" in the October 1939 issue of *Minnesota Medicine* by the Honorable J. A. A. Burnquist, World War I governor of Minnesota and later attorney general of outstanding ability; *Invited and Conquered* by J. Arthur Myers, M.D.; and traditional and miscellaneous information.

Ninety Years of Obstetrics and Gynecology

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TO TRY TO TELL the story of the last ninety years of medicine is to attempt to condense almost the whole background of our present understanding. This would require a series of books rather than a single paper. There were giants in the land prior to 1870—but they were too few and had too little encouragement and too few facilities. Too many stumbling blocks of prejudice, both legal and popular, were put in their way. Of decisive importance, however, was the lack of basic information in other fields of science.

Medicine has always been peculiarly dependent upon the development of the basic sciences and has had to wait until scouts from these fields opened the way before it could move forward on the newly won ground, consolidate itself, and extend the new information to practical human purposes. No exact date can be set as the beginning of this, since the process was gradual, with an accelerating velocity. But if one were required to choose a date at which the development of obstetrics and gynecology might most logically be said to have begun this acceleration process, it would probably be wisest to choose the period between 1850 and 1870. This is probably true of all medicine, and it is interesting that *THE JOURNAL-LANCET* spans the period since that time. This period of beginning for obstetrics and gynecology has been chosen because 1850 or, if one wants to be more exact, 1847 marks the publication of the work of Semmelweis on puerperal infection; 1851 marks the appearance of Litzmann's publication of the work of Michaelis, three years after the latter's death, which gave us in a single contribution the majority of the presently used clinical information about the obstetric bony pelvis.

During the period 1850-70, there was no gynecology worthy of the name. This had to wait for the twentieth century and the development of an understanding of ovarian function, recognition of the details of the menstrual cycle,

establishment of safe surgery, and a host of other things. Obstetrics was, of course, old, but it was still in the hands of midwives whose only interest lay in practical problems. Basic knowledge was missing, and the midwives were in no position to supply it.

In the late eighteenth and early nineteenth centuries, England and France were well in the lead. The obstetric forceps had been introduced by Chamberlen and gave the physician, with his lack of knowledge, a tool with which to cause trouble. One should read Laurence Sterne's *Tristram Shandy*, written about 1750, for an amusing description of Dr. Slop and his misuse of the forceps. It is not generally known that in Germany at this time version and extraction were widely applied by the physician for most difficulties of delivery, real or imagined, when a midwife was so rash as to call him. Both doctors and midwives taught the student midwife, and so the blind led the blind.

Out of this welter in Europe came a number of men at the end of the eighteenth century. One of the major influences was exerted by Johann Boer, who worked in the complicated academic circumstances of Vienna. Among other things, he had a school of "natural obstetrics," which had a far-reaching influence on the continent. His work had much to do with encouraging thoughtful physicians to enter the field of the study of obstetric problems and set the tone for later work.

The two important contributions which appeared about 1850 were those of Michaelis and Semmelweis. These men changed the whole course and pattern of obstetrics, and a word should be said about them and their work.

Michaelis took over the direction of the University Women's Clinic in Kiel, Germany, from his uncle, Wiedemann, in 1825. He set out to study the obstetric pelvis, about which almost no information existed. His handwritten clinical notes are available in a beautiful copperplate script which is so small that magnification is necessary to read it. The observations and measurements are astonishingly detailed. There is little of the clinical aspects of pelvic dystocia,

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which has been added since. Unfortunately, Michaelis became depressed in association with the death from puerperal sepsis of a patient who was also a relative, and, in 1848, he committed suicide. His work was completed and published in book form in 1851 by his successor, Litzmann. It is a book which all students of obstetrics should read.

The story of Semmelweis is more generally known. His main work was done in the first Women's Clinic in Vienna, where he recognized that the horrible mortality rates from puerperal infection were the result of something which was introduced by the hands of the physicians who examined the women in labor. The clinic where he worked was used to train local and foreign students and physicians. The average mortality rate in this clinic for the year 1846 was 13.7 per cent, and almost all of this was due to puerperal infection. In May 1847, Semmelweis introduced careful hand washing with various compounds, and for the year 1848, the mortality rate dropped to 1.27 per cent.

The bitterness and jealousy which followed are difficult to understand. The medical school faculty was impressed with the broad significance of the findings and appointed a committee to look into the matter. Klein, the director of the Women's Clinic, refused to allow the committee access to the clinic and may well have postponed for twenty years the beginning of bacteriology and transferred this honor from Vienna to Paris. Whatever his contributions, and they were considerable, he will always be remembered for this one unfortunate decision.

Semmelweis died in 1865 of septicemia—his own disease, so to speak—secondary, however, to general paresis. His work changed the whole course of obstetrics.

From this point, the German school took over the lead in obstetrics, as in other fields of medicine. This was due in large part to a system which carefully chose superior men on a highly competitive basis, freed them for study, and supplied them with the circumstances in which such study could be done. The medical world will always be indebted to the German university system. Using this system, the United States began to compete seriously in the 1920's and finally took over the leading position just before World War II. The enormous significance in both directions of a political system upon the development of science is worth serious thought.

Development of gynecology had to wait for a number of things. The basic control was and still is histologic. Hitschmann and Adler, in 1912 in Vienna, finally recognized the details of the

menstrual cycle. Edgar Allen and Doisy in this country recognized the vaginal cycle in the mouse which opened up the field of ovarian endocrinology. Corner and Willard Allen in Rochester, New York, isolated progesterone; Allen dropped and spilled the whole world's supply of this in a single vial, but plenty more was soon available.

The group at the Carnegie Laboratory in Baltimore—Streeter, Hartman, and many others—made the primate a useful and controllable tool, so that accurate early embryology and many aspects of endocrinology were soon available. Markee learned to transplant endometrium to the anterior chamber of the animal eye, permitting direct observations of response. Reynolds, also working in the Carnegie Laboratory, established an interest and understanding of the physiology of the pregnant uterus. It is said that the first primate egg which was recovered was by far the world's most expensive, but, in retrospect and as related to the significance of the contributions made by the group, it was a very cheap egg indeed.

Running like a golden thread through all of this was the work of Robert Meyer, who worked in the first Women's Clinic in Berlin and spent his last years in the Department of Obstetrics and Gynecology at the University of Minnesota Medical School. He began an enormously industrious career just before the present century. For most of his life, he was the stable control to whom everyone turned for confirmation and help. His career coincided with the real beginning of the application of histologic techniques to the understanding of gynecologic problems. If one person were chosen who had contributed most over the broad field to the development of gynecology, he would be the author's candidate for that honor. His specific contributions are too many to list, but mention should be made of his work in early embryology; the development of the generative tract; the changes of early malignant disease; the ovarian tumors, which are largely his own contribution; and, perhaps most important of all, the training, stimulation, and help which he gave over a long career to so many, who in turn made their lesser contributions.

Gynecology also had to wait for separation from general surgery. Not until it was divorced from surgery did it achieve the study and attention which it deserved. The surgical techniques were not difficult, but the general surgeon thought of gynecology only in terms of operative methods and made no contributions of significance to the field. For separation of gynecology

from surgery, one must again thank the German school that developed the Women's Clinics. The concept was rapidly accepted in this country, to the great benefit of the discipline.

Many more features of development and many more contributions from individual men could and should be listed if space were available. It remains now to consider the present states of the disciplines and what the future promises.

Real progress in the understanding and the application of this understanding in obstetrics is clear. In the United States, maternal mortality rates have dropped in the past thirty-five years from between 5 and 6 per 1,000 live births to about four-tenths. In Minnesota, this has dropped to about two and a half tenths in terms of so-called obstetric deaths. Only about 10 per cent of these deaths are preventable, as determined by a really detailed study of each maternal death. This has to be compared to a 75 per cent preventability only eighteen years ago. The apparently irreducible minimum maternal mortality rate is a little less than two-tenths per 1,000 live births, and Minnesota is approaching that figure. Remaining problems are now fairly well pinpointed.

Real progress has been made in developing physicians who practice in obstetrics the concept that they must control and use all of the technics of patient work-up and study which are available. The time has long since passed when an obstetrician could work effectively with only a knowledge of obstetrics. He has been taught and is more or less effectively using the tools of the internist. This has been a basic reason for progress in the field.

Gynecologic problems are now well understood, and methods of objective diagnosis are well established. There is much more work to be done, as is, of course, true in all fields of medi-

cine. The difficulty in gynecology is that there are no objective measures of the efficiency of the application of available knowledge to the general public, as there are in obstetrics. Some sort of study of this is urgently needed. Only in the field of gynecologic malignant tumors is it possible to express efficiency accurately. In this area, splendid progress has been made, and these tumors are probably better understood and more effectively handled than are tumors in any other anatomic area. This is not to say that there is any satisfaction with presently available results. But, bit by bit, the situation is improving. One would have been rash indeed to have prognosticated twenty years ago the remarkable improvement in the cure rates for carcinoma of the cervix, of the endometrium, and of the vulva or the striking change in the outlook for chorioepithelioma.

This leaves a major problem which has scarcely been more than explored. Very little is known about the basic circumstances of the intrauterine fetus. This involves complicated biochemistry. Investigations are under way, and there is good reason to believe that information obtained will have important practical applications. In all probability, this is the important field of study for obstetrics and gynecology in the immediate future.

In very brief form, these are some of the things which have gone on in the field of obstetrics and gynecology over the life span of *THE JOURNAL-LANCET*. They have been stirring years and can certainly be said to encompass the major development of medical knowledge. It would be rash to expect that the increasing velocity of growth of medical knowledge could continue at this rate, but it probably will. How fascinating it would be to have a peek at the pages of *THE JOURNAL-LANCET* after another ninety years!

ELECTIVE INDUCTION of labor in patients in whom the cervix is ripe may shorten labor and minimize the patient's discomfort.

When the cervical os begins to open, permitting finger examination about seven to ten days before the predicted delivery date, the cervix is said to be ripe. Labor may be induced in about 50 per cent of patients at this time.

The cervical os is examined with two fingers, using pHisoHex as a lubricant. After the membranes have been gently removed from the lower uterine segment, a long Allis hemostat is inserted between the two fingers. A slight twisting motion is usually sufficient to rupture the bag, the tear then being enlarged by the fingers to allow the escape of amniotic fluid. In most patients, a uterine contraction will occur within thirty to sixty seconds, followed by active onset of labor in about forty-five minutes.

For 745 patients with spontaneous onset, the usual length of labor was about nine hours, whereas the time was shortened to approximately five hours in 410 patients in whom labor was artificially induced.

J. L. JACKSON: Elective induction of labor. *Texas J. Med.* 55:956-960, 1959.

Industrial Medicine—The Changing Scene, 1870-1960

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INDUSTRIAL MEDICINE finds its roots in ancient history. Developments of the last ninety years should be viewed in the light of its earlier history and the concurrent advancement of industrial hygiene together with changing social attitudes and economic influences. From fragmentary beginnings, industrial medicine and hygiene in America grew slowly during the earlier years and, at times, rapidly under the stimuli of two world wars and the technological revolutions within various segments of industry. No attempt has been made in this limited review to include the names of the many pioneers, distinguished leaders, educators, and research scientists to whom tribute is due. The names that do appear are those whose writings have been quoted or whose names are linked with some of the milestones of progress.

HISTORY

Some knowledge of occupational diseases and of health risks at work was available dating back to the writings of Aristotle, Plato, Hippocrates, Galen, and others in Greek and Roman times. Postural deformities among artisans, dangers of handling sulfur and zinc, toxic properties of lead and mercury, and observations on the hazards of work had been recorded in the earliest literature.

During the fifteenth and sixteenth centuries, the first of the early publications on industrial hygiene appeared, including reports on the toxicity of carbon monoxide, nitric acid and heavy metals, and the first observation with reference to silicosis. An Italian physician of the seventeenth century, Bernardino Ramazzini, acclaimed the father of industrial medicine, was a great teacher and scholar who wrote of the occupational diseases among tradesmen and artisans of the time. His treatise, *Diseases of Workers*, became the basis for the extensive literature on industrial hygiene of modern times.¹ His originality, powers of observation, and ability to re-

late occupations and disease are honored to this day by a society of industrial physicians and publications bearing his name.

The rise of industrialization beginning in the late half of the eighteenth century, with England leading the way, brought with it, belatedly, society's and government's concern with the wretched working conditions in mines, mills, and factories. Donald Hunter's² *Diseases of Occupations* contains an excellent account of the accident and health hazards of the early machine age. Social reform measures were undertaken by legislation, and the early laws of England, France, Switzerland, Germany, Austria, and Belgium provided a concept and ultimately a basis for enactment of Workmen's Compensation laws during the twentieth century. They also marked the beginning of national and state efforts to promote and regulate industrial hygiene.

The first recorded treatise on industrial dermatology appeared in 1806, the work of a French dermatologist, J. L. Alibert. The year 1821 marked the beginning of the French periodical, *Les annals d'hygiène*, which did more for the promotion of industrial hygiene than any other publication of the period.³ The earliest statistics relating to occupational mortality and morbidity appeared in 1840. The great French physiologist, Claude Bernard, is said to be the first who, in 1865, fully appreciated the scientific significance of the investigation of poisons and directed his attention particularly to curare and carbon monoxide.⁴ His important concept is quoted as follows: "Poisons can be employed as agents of life's destruction or as means for relief of disease, but in addition to these universally recognized uses there is a third that particularly interests the physiologist. For him the poison becomes an instrument that dissociates and analyzes the most delicate phenomena of the living machine, and by studying attentively the mechanism of death in diverse types of poisoning he can learn indirectly much about the physiological processes of life."

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Contributions of the German-speaking countries during the nineteenth century included the work of E. von Bibra and L. Geist in 1847 on phosphorus poisoning of matchmakers. In 1845, Halfort described the pathology following exposure to toxic gases and dusts and the prevention of disease associated with faulty posture, fatigue, and abnormal temperatures. Hirt's work, published in 1870, included an extensive treatise on occupational diseases.

A review of the occupational health publications in the United States prior to 1900 was published in 1955 by Dr. C. P. McCord,⁵ consultant, Institute of Industrial Health, University of Michigan. Over 200 titles are listed, excluding articles of a general nature, such as those with reference to tuberculosis in industry, hours of labor, need for legislation in worker protection, and so forth. Among the early American contributions was Dr. B. W. McCready's prize dissertation in 1836, sponsored by the New York State Medical Society, entitled "On the Influence of Trades, Professions, and Occupations in the Production of Disease." It described the unhealthy working conditions of seamen, women and children in textile factories, tailors, shoemakers, printers, butchers, smiths, goldbeaters, carpenters, and so on. A general discussion of the effects of coal mining on health appeared in *Transactions of the Medical Society of Pennsylvania* in 1869 by J. T. Carpenter.

The Industrial Revolution in the United States was in full stride by the year 1870 and reconstruction following the Civil War brought industrial expansion at a rate unprecedented in history. The first transcontinental railroad had been completed, oil had been discovered in Pennsylvania, new cotton mills in the South were competing with New England, the rise of tobacco manufacturing had started, and oil refining on a mammoth scale was undertaken by John D. Rockefeller. Industrial development and westward expansion were aided by the stream of immigrants who flowed into the United States with no retarding legal restrictions. In the decade between 1870 and 1880, well over 2,000,000 immigrants came from Europe. The population of the country was 38,500,000 and about one-third of the people were dependent upon industrial employment.⁶

An early law to regulate industrial employment in the United States prohibited child labor and was passed in Pennsylvania in 1837, and another, limiting women's work to ten hours a day and six days a week, was passed in Massachusetts in 1874. The first federal statute relat-

ing to labor was enacted in 1875. The right to enter factories was granted to inspectors, and the first regulation for industrial dust control was passed in Massachusetts in 1877. Federal legislation for railroad safety was not enacted until 1893, but articles on the relationship of color blindness to railroad and maritime accidents appeared as early as 1878. The Swedish physiologist, Alarik F. Holmgren, devised his wool skein test for color vision of railway employees in 1876.⁷ Thereafter, tests for color blindness became obligatory in the railway services of most countries.

The date of 1870 has been offered as marking the close of the revolution in biologic sciences. It was the completion of a movement that brought the natural sciences into biology and with the biologic revolution, surgery started to move forward. The discovery and acceptance of general anesthesia in the 1850's introduced a new era, followed by measures to control infection with the application of Pasteur's concept and the contributions of Joseph Lister in 1867.⁸ Industrial medicine in America, according to some writers, had its genesis in the surgery of trauma.

The railway surgeons were the first organized industrial physicians in the land, and, in 1888, the American Association of Railway Surgeons was founded. Beginning with about 200 members, its membership had grown to more than 3,000 when the history of this organization was reviewed sixty-five years later in *Industrial Medicine and Surgery*.⁹ This monthly journal had earlier become the official publication of the American Association of Railway Surgeons. Each year since its founding, the association has held annual meetings, including a scientific program for presentation of papers on subjects in occupational medicine as well as surgery.

The period between 1870 and 1900 was one of large scale activity in bridge and underwater tunnel construction in America, and a score of papers were published during this time on the physiologic effects of compressed air. One of these describes 119 cases and 14 deaths from caisson disease during the construction in 1871 of a bridge over the Mississippi River at St. Louis.¹⁰ The first American reference to caisson disease appeared in an article of 1843 describing the use and effects of compressed air in mining.¹¹ Dr. R. T. Legge records that E. W. Moire in 1904 was the first to use a medical air lock for recompression of underwater workers during construction of the Hudson River tunnel.¹² Within a few years, state laws were passed requiring

physical examinations for persons working in compressed air.

PNEUMOCONIOSIS

During a span of three years, 1869 through 1871, the medical literature of the world on pneumoconiosis, including silicosis, was increased by 14 publications, according to a bibliography compiled in 1933.¹³ The subjects are indicative of the broad interest at the time in pulmonary dust diseases and their control—studies on pathology, dust sampling, and the relationship of many occupations, including mining, shell cutting, and clay working, to tuberculosis. At the beginning of the twentieth century, following the invention of radiology and its application, there were great advances in the diagnosis and case study of pulmonary dust diseases. It is of interest to note that this bibliography includes 2,768 references on this single subject beginning with Agricola's *De re metallica* in 1556 and concluding in 1933 with a booklet on silicosis by Dr. R. R. Sayer which was published by the United States Public Health Service. Publications on this subject have continued to appear since then at a high rate, with the contributions of industrial hygienists and engineers adding greatly to the knowledge of principles and safe practices in the control of dust.

Current concepts of pneumoconiosis were reviewed in June 1959 by Dr. Paul Gross of the Industrial Hygiene Foundation, Mellon Institute, Pittsburgh.¹⁴ Earlier concepts which held that "inert" dusts, such as dusts of soft coal, kaolin, aluminum oxide, and silicon carbide, were harmless are being revised. Recent experimental evidence indicates that such dusts are capable of causing a massive pulmonary fibrosis in association with tuberculosis. This work followed observations among miners that soft coal dust may cause severe obstructive emphysema, often terminating in decompensated cor pulmonale.¹⁵ The older term, "tuberculosilicosis," should be abandoned in favor of the more inclusive, "tuberculo-pneumoconiosis," as proposed by Gross. The mechanism of fibrosis has been a subject of speculation, particularly in silicosis and asbestosis, for which there has been a succession of theories. The most recent postulates that silica crystals possess surface activity which denatures proteins and that the deposition of beta and gamma globulins in the characteristic nodules of silicosis indicates an antigen-antibody reaction. Differences between nodular and diffuse fibrosis, which were studied by Leroy Gardner¹⁶ during the time he made his great contributions,

were believed to be due to the adequacy of lymphatic drainage. This explanation is not acceptable in the light of recent studies of silicosis, thus reopening other areas for additional research.¹⁷

LEAD POISONING

Lead, said to be the oldest of all industrial hazards, has been the subject of the most extensive literature. Dr. McCord published a series of 7 articles during 1953 and 1954 on "Lead and Lead Poisoning in Early America."¹⁸ He records that the early explorers along the upper Mississippi River, including Robert de La Salle, Louis Hennepin, and Le Sueur, had an interest in the lead deposits at the present site of Galena, Illinois, where lead mining apparently had been attempted in 1687. Of historic interest is Benjamin Franklin's knowledge of lead poisoning, said to be greater than that of many physicians of his time.

Industrial toxicologists¹⁹ give credit to Dr. Alice Hamilton²⁰ for her work before World War I in uncovering the extent of lead intoxication among American workers. Her reports on the surveys she conducted in numerous other industries and her many publications are classics in the literature of occupational medicine. The occurrence of lead intoxication has steadily decreased, and, today, serious poisoning of industrial origin is rare, thanks to the science of industrial hygiene and methods for early diagnosis. Because lead is a cumulative, insidious poison and is so extensively used in industry, there was controversy over its mode of entrance into the body, its absorption, its storage, and its excretion. Several of the issues were settled by "Lead Studies" conducted at Harvard between 1922 and 1924 by Aub and his colleagues.²¹ The basophilic aggregation test for lead intoxication was discovered by McCord in 1924. In 1933, the United States Public Health Service established maximum permissible concentrations of the lead content of the air to which workers are exposed.²² The relative toxicity of lead and its compounds, the solubility of various lead compounds in blood serum, and the excretion of lead in the urine have been studied and reported by Fairhall²³ and Fairhall and Sayers.^{24,25} Clinical and laboratory investigations and extensive studies of the metabolism and excretion of lead have been carried out by many investigators, the work of Kehoe and Aub being outstanding.

The fundamental toxic action of lead on tissues is not understood, although it is said to inhibit certain enzyme systems. Symptoms of

intoxication are more severe than can be explained by microscopic examination of tissues. With exposure, the amount of lead absorbed is estimated from the amount in the urine or blood. Insignificant amounts are normal, but higher levels may be diagnostic or predictive of intoxication. Porphyrin excretion has been used to determine the likelihood of lead intoxication, but the absorption of several other metals may cause excretion of toxic porphyrin. Thus, the exposure to lead must be definitely established in the interpretation of this test.²⁶ A screening technic has been developed making use of the lowering of diastolic blood pressure and increased pulse pressure as an index of impending intoxication.²⁷ Tests at the Haskell Laboratories, Wilmington, Delaware, indicate that this offers a useful technic for checking exposure to several toxic chemicals.

The organic compound, tetraethyl lead, used in gasoline for its antiknock properties, is readily absorbed through the skin in addition to being absorbed from vapor in the respiratory system. Effective control measures are provided by the manufacturers and distributors of the lead additive, including periodic tests for absorption conducted on the workers who may handle it. The control program is a result of a cooperative agreement made in 1926 between industry and the United State Public Health Service. The magnitude of this program is appreciated when one considers the tremendous quantities of ethyl gasoline used in today's automobiles. The lead exposure associated with handling and dispensing it at service stations in the United States is negligible.²⁸ However, there have been cases of lead poisoning reported among workers who recondition automobile engines in which there are lead deposits.

The age-old problem of lead poisoning, recognized since the time of Hippocrates, continues to be a field for investigation and study. Outstanding research has been accomplished, but problems in toxicology, diagnosis, and treatment remain. Fortunately for industry, the practices and technics for prevention of lead absorption are well developed and, if expertly applied and rigorously maintained, afford good protection.

PHOSPHORUS POISONING

The history of phosphorus poisoning in the match industry during the late nineteenth and early twentieth centuries is cited by historians to demonstrate society's indifference toward problems of industrial hygiene. The first American case of phosphorus necrosis was reported in

1851, according to Dr. Hamilton in her autobiography, although "lucifer" matches had been manufactured in the United States for about fifteen years. Severe cases of necrosis of the mandible, or phossy jaw, had appeared in 1844 among the workers of the first match factories in Germany and Austria, twelve years before legal steps were taken in the interest of prevention. Elsewhere on the continent, in England, and in America no action was taken until another fifty years and more had elapsed. Phosphorus necrosis was minutely described in articles published in America in 1854, 1856, and later, according to the review of the early literature previously cited. The poisoning was slow in onset, the average time being five years after exposure to the phosphorus fumes, and as high as 4 per cent of the workers exposed were victims of the disease. Sanitary precautions, no matter how rigorous, could not reduce the hazard to a degree that was "safe." In 1898, a harmless substitute, sesquisulfide of phosphorus, had been discovered and soon became available, but six more years elapsed before the first of the governments of the match producing countries prohibited the manufacture and sale of poisonous phosphorus matches. When Dr. Hamilton first became interested in this occupational disease in the early 1900's, she confirmed its extent and severity in the United States and, what was more disturbing, found indifference to the hazard on the part of industry and little concern on the part of the medical profession. Prevention was not accomplished in the United States until 1913 after Congress imposed a prohibitive tax on poisonous phosphorus matches.²⁹

The history of phosphorus necrosis—in this case an occupational disease—is a striking example of the delay in acquisition of medical knowledge and its application. Without public awareness of health hazards and cooperation of industry and government, progress based on medical science may be needlessly delayed.

NEW DEVELOPMENTS

Fortunately, with the advent of the twentieth century, a new interest in the health and welfare of the American worker developed. In the decades that followed, public and private organizations were founded dedicated to the conquest or prevention of disease and accidents; to the support of research in industrial hygiene and occupational diseases; and to the establishment of additional educational resources and training programs for physicians, nurses, hygienists, and safety engineers associated with in-

dustry. The growth, increasing number, and strength of these organizations in this as in other fields for health and welfare promotion added greatly to the progress of industrial hygiene, accident prevention, and occupational disease control.

Following enactment and extended coverage of Workmen's Compensation laws, industry learned that preventive measures and early detection of disease were much less costly—in terms of good will as well as dollars—than the care and disability expenses of occupational disease and injury. Originally, having been alerted by expediency to its role in the field of health, the value of specialized medical services and readily accessible facilities became apparent to management. Larger corporations and those obviously having occupational disease hazards undertook case-finding programs and studies of industrial health problems. Research laboratories and establishments for environmental study were equipped and staffed by some corporations and others joined together in supporting such establishments, usually associated with large universities. Prevention of silicosis, an early and major concern of mining companies and many other segments of industry, initiated this movement. The vast and complex chemical industry, including petroleum, plastics, and synthetics, found practical knowledge of toxicology to be indispensable. Hazards of ionizing radiation and of exposure to excessive noise levels have been added to the older problems of harmful dusts, toxic fumes, and the handling of skin irritants or sensitizing materials. Industrial carcinogenic agents has become a special field of study. Industry plays a major role in sponsoring and financing the development of general and specialized knowledge of occupational diseases and in making practical its application.

The American Medical Association has had a continuing and active interest in preventive medicine since 1859. In that year, a section was formed which, over the years, had a succession of names, including, among others, hygiene, sanitary science, industrial medicine, public health, and preventive medicine. The section took a prominent part in gaining recognition for preventive medicine and occupational medicine. In 1952, the American Board of Preventive Medicine was incorporated as a medical specialty with the authority for certification in the specialty of public health in 1952, in aviation medicine in 1953, and in occupational medicine in 1956.³⁰

The Council on Industrial Health is the

official agency of the American Medical Association designated to serve the medical profession at large in respect to developments in this field and in industrial medical relations. The first steps were taken toward its formation in 1936 when several problems were brought to the attention of the association's House of Delegates, including the chaotic nature of Workmen's Compensation administration, the need for better clinical and administrative management of occupational disease, and the need for better recording and dissemination of information on industrial dermatitis. In more recent times, the council has directed its attention to better intra-professional communication and understanding of industrial health problems and services, to better opportunities for industrial physicians to serve in this field, to improved professional training, and to cooperation by local and state medical societies. The council issues reports from time to time, including those describing the basic aims of industrial health, the physician's place in industry, and the administration of an industrial medical program.³¹

The American Medical Association publishes the monthly journal, *Archives of Industrial Health*. It is also the official publication of the American Academy of Occupational Medicine, an organization of full-time physicians in occupational medicine.

The Industrial Medical Association, with headquarters in Chicago, was organized in 1915. In the early years of the association, several of its officers were officers or prominent members of the American Association of Railway Surgeons.³² Until 1955, it was known as the American Association of Industrial Physicians and Surgeons. In 1920, it had 400 members; today it has a membership of about 4,000 in the United States, Canada, Latin America, and Hawaii. The monthly journal, *Industrial Medicine and Surgery* served as the official publication of the association until January 1959, when the association launched its own publication, the monthly *Journal of Occupational Medicine*. Since 1945, the association's annual convention has been held concurrently with the meetings of 4 other national organizations having related interests—the American Industrial Hygiene Association, American Conference of Governmental Industrial Hygienists, American Association of Industrial Nurses, Inc., and American Association of Industrial Dentists. The association is organized on a geographic basis, comprising state districts or component societies. The Minnesota Academy of Occupational Medicine and Sur-

gery, founded in 1954, is such a component, with a membership of about 85 Minnesota physicians.

The Occupational Health Institute, Inc., is an educational organization created by the Industrial Medical Association to promote the cause of health in industry. Since 1951, the institute has conducted the program of evaluation, approval, and certification of medical services in industry on the basis of standards established by the parent organization.³³ Until then, this program had been conducted by the American College of Surgeons. Following appointment by the college of a Committee on Industrial Medicine and Traumatic Surgery in 1926, the college started this activity in 1932.

The scope of this paper does not permit an account of the major contributions made by industrial hygiene engineers, toxicologists, and hygienists working as private consultants, as employees of industry, or in government service. Without their knowledge and skills, there would have been no occupational disease control programs as we know them today. In all industrial medical departments, beginning with the earliest, the day to day success of the employee health service has depended in large measure upon the special knowledge and talents of industrial nurses. Recent arrivals on the scene include industrial psychiatrists and psychologists. Dermatologists, ophthalmologists, otologists, and roentgenologists are specialists closely associated with industry who have contributed very substantially, and their work is well represented in the occupational disease literature.

The history of government activities in advancement and support of industrial medicine and hygiene, under the leadership of the Public Health Service, is a story in itself. Physicians, hygienists, and toxicologists in government service have been key figures in all phases of development. To the Division of Occupational Health of the United States Public Health Service goes the credit of developing the epidemiologic approach to occupational disease. Industrial hygiene research laboratories and facilities for analytic studies, together with consultant services, are maintained by many state health departments as well as the Federal Health Service.

COMMENT

The nation's industrial potential and productivity are greater forces than ever before in controlling the economy, shaping the social system, and determining national security. All elements of our society, including the medical profession,

have been and will continue to be profoundly affected by the needs and problems of industry. It goes without saying that the needs are constantly changing and increasing with advances of technology, expanding production, and the growing size and complexity of modern industry.

Tremendous progress in clinical medicine has been made during a ninety-year period of sweeping industrialization, and today American medicine leads the world in research and training at its clinical centers and in the practice of scientific medicine. The great benefits of this contribution to society and industrial prosperity are beyond reckoning.

Industrial medicine has grown within the framework of organized medicine and is now a well-defined, vigorous field of practice and specialization. In one respect, this is a recent development, although industrial medicine had long been known and many American doctors identified with industry during the last sixty years and more have been prominent members of the profession, representative of several specialty groups. Further definition and the pattern for future growth came in 1956 with the formal union of preventive medicine and industrial medicine culminating in recognition as a medical specialty. Under this concept, preventive medicine in industry, more accurately called occupational medicine, draws heavily upon the bodies of knowledge and disciplinary resources alike of preventive medicine, clinical medicine in industry, and the medical aspects of environmental hygiene.³⁴ With its heritage, enlightened concept, and important mission, the future for this field of medicine, by whatever name—industrial medicine, occupational medicine, or preventive medicine in industry—is unlimited.

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PARTICLES OF glass dust are not toxic and do not irritate the skin and mucous membranes. Because glass is insoluble, it is biologically inert, and the particles tend to present flat surfaces rather than sharp edges to tissue, causing them to float upon the film of any fluid.

In rabbits, blinking was found to clear glass dust particles from the ocular conjunctiva, preventing corneal defect.

Ingestion of glass flakes by rats does not cause toxicity or trauma to the gastrointestinal tract. When rats were given a diet consisting of equal parts of ground glass and food, a slight depression in growth rate occurred, due entirely to a decreased intake of food caused by the bulk of the ground glass.

Pulmonary changes in guinea pigs and rats exposed to fine glass dust for one year and in rats given intratracheal injections of the dust consisted only of small focal alveolar dust cell collections free of fibrosis. Focal intracellular glass dust deposits in the pulmonary lymph nodes were also not associated with reactive fibrosis. The dust does not cause bronchial disease.

Fine glass dust produces a more benign pulmonary effect than does kaolin dust because the stored dust remains alveolar rather than occupying interstitial positions and is therefore easier to clear from the lung.

Coarse glass dust or flakes injected into the lungs of rats cause alveolar cell proliferations and a thin investment of the fragments by a single layer of flattened cells and a few delicate collagen fibers.

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History of the University of North Dakota School of Medicine

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University of North Dakota School of Medicine.

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"THE UNIVERSITY OF NORTH DAKOTA does not have what can be called a medical school." This statement was part of the report of the accrediting agencies in 1936 after the survey committee had visited the school. It had noted the cramped space, the lack of a medical library—what books there were, were housed in an alcove of the main library—the lack of research, the dearth of equipment, and the minuscule budget. It also noted, however, with some incredulity, that the school did manage to graduate students each year who transferred and successfully completed their medical education elsewhere. A look at the subsequent careers of the graduates of the five years immediately preceding this vitriolic report is interesting and informative. In this period, 142 graduates transferred. Of these, 32 now hold certification by the American Specialty Boards, an *additional* 20 successfully passed the National Board examinations, and 11 of this group now hold teaching appointments on medical school faculties of schools such as Harvard University, Columbia University, University of Minnesota, Duke University, University of California, College of Medical Evangelists, University of Chicago, University of California at Los Angeles, and the University of Washington. The remainder are in practice.

Surely these were not graduates of this medical school. Surely there must be some mistake. There was. The mistake was in trying to measure teaching by measuring floor space, gauging teaching by faculty publications, estimating teaching ability by the number of degrees and the salary paid to members of the faculty, and evaluating the library by the number of books, not those read. Medical education is an intellectual process and cannot be measured. The survey committee could not measure and did not appreciate the virtuosity of a small group of dedicated teachers and students who knew that they could not coast into an education. The facts speak for themselves.

Begun as a basic science, two-year school of medicine, in common with 9 other similar mid-western schools, both North and South Dakota have been maintained as such, whereas the others have expanded into four-year schools. There are no large centers of population in this state of large area. Therefore, there is no concentration of patients to provide for clinical teaching. There are no plans in the immediate future for expansion to a four-year school.

The Medical School of the University of North Dakota has, since its beginning, been an integral part of the University and, as such, has suffered the fortunes of the University and the state.

In 1883, the Dakota Territory, not to become a state until 1889, was opening up as the last frontier of the farmer. In this year of 1883, the last spike was driven in the Northern Pacific tracks in Montana, opening up at last the rich farming lands of the Red River Valley and, later, the range lands of the western part of the state. The area was booming—only thirteen years before, the population had been 2,045, increasing to 36,909 in 1880 and to 116,000 in 1885. Into this area of open prairie came boom towns, complete with stores, town halls, and saloons. The earliest settlers were from the East and eastern Midwest, later matched by equal numbers of immigrants from Norway, Sweden, and Germany.

In this pioneer setting, the University of North Dakota was founded in an open wheat field 2 miles from Grand Forks, a city at the time of 3,000. In 1887, the idea of a medical school was promulgated, and the legislature actually appropriated \$1,000 of a total University appropriation of \$44,000 for its support. A change in administration removed the chief proponent, and the idea died—perhaps a justifiable demise—since there was but one building on the campus to house the entire University—faculty offices, chapel, classrooms, library, laboratories, and part of the living quarters for students.

In 1902, the opening of a school of pharmacy was announced, which really served to build up the premedical curriculum, and the faculty voted in 1905 to establish a medical school. Melvin Brannon, professor of biology and curator of the museum, although possessed of only a master's degree from Wabash College in 1890, had initiative and organizational ability and not only planned wisely but enlisted the firm support of the physicians of the state and the interest of the legislature. The State Health Laboratories were to be an integral part of the school. Mr. Brannon took an active part in the development of a local water purification plant—one of the earliest in the nation—following a typhoid epidemic in 1893 and, at one time, was also director of athletics. Dr. J. D. Taylor of Grand Forks took up the cudgel for the medical school, enlisting the support of the local physicians and pushing a law through the legislature to make cadavers available to the new school. Plans were made after Mr. Brannon had conferred with other medical school deans and educators, and classes were started in 1907, and a class of 1 was graduated in 1909. The faculty at that time consisted of 3 full-time teachers: Mr. Brannon; Dr. Archie McDonald, a graduate of Johns Hopkins University, who taught anatomy; and Dr. Gustave Ruediger, a graduate of

Rush Medical College, who served as pathologist and bacteriologist for the State Health Laboratories as well. The school was housed in one of the top floors of the newly erected science building, adjacent to the State Health Laboratories and remained there until the present medical school building was built in 1949.

Equipment was minimal, the budget was small, and faculty and students were few. However, from the start, there was evidence of the recognition of sound teaching, noted by Abraham Flexner a few years later. The school maintained a close connection with the State Health Laboratories to their mutual advantage. For many years, they were administratively united and, even now, are housed in the same building. Provisional approval was given by the American Medical Association and the Association of American Medical Colleges in 1907.

In 1909, Flexner visited and surveyed the school as part of his survey of American medical education. North Dakota fared well in this report, which is filled with blunt and scathing comments on numerous medical schools, many older, larger, and wealthier than North Dakota.

He noted the attendance of 9 students, a teaching staff of 9 professors and 7 instructors taking part in the work of the department, the budget of \$6,300, and the income from fees of \$450. Under laboratory facilities he noted: "The laboratory of bacteriology, being at the same time the Public Health Laboratory of the State, is well equipped and very active. Subjects given in the regular university laboratories are likewise well provided for. For the specifically medical subjects—physiology, pathology, and anatomy—the provision is slighter. The students are, of course, few. A library and a museum have been started." His conclusion: "The two Dakota schools have taken time by the forelock; before any vested proprietary interest could be created, they have fixed the State practice requirement at two years of college work. . . . The State, though thinly settled, is prosperous, and no anxiety is felt that the high standard will deplete the medical profession of the State. . . ."

A keen observer and analyst, even Dr. Flexner could not foresee the years of financial stress that were to plague the University and threaten its very existence in this state where so much of the land is marginal in rainfall, and a dry year can cut the state income in half and a drop in the small grain market to a third.

In 1911, faced with the need for an administrator with more qualifications and equipped with more than a master's degree and after a crop failure in 1910, the president seized upon

the resignation of Dr. McDonald and the resignation of the dean of liberal arts to make a change that was to become the most important in the history of the school. Dean Brannon was made dean of liberal arts, and Dr. Harley E. French, professor of anatomy at South Dakota, was appointed professor of anatomy and dean.

Dr. French was a midwesterner and knew the area and its problems well. He received his medical degree from Northwestern after teaching school for several years. He had not only a well trained mind but a classical education and was possessed of tireless energy and absolute integrity. Subsequent years in one of the longest deanships in medical history were to put him to the test. From his appointment in 1911 to his retirement in 1947, Dr. French faced, and at times faced alone, almost insuperable calamities, which culminated in the thirties.

Confronted with a minuscule budget, as low as a biennial appropriation of \$21,000 in 1933, and an absolute minimum of space and equipment, Dr. French deliberately concentrated his efforts on teaching, deferring research because of necessity. His faculty consisted of a hard core of dedicated teachers who maintained a teaching program which can perhaps best be documented by a look at the product—the graduates. For years, Dr. French patiently presented the needs of the school to the people and the legislature, and since the state was always without the income, his requests were turned down. He held off the accrediting agencies and transferred student many times on the strength of his integrity, which was recognized by his peers.

The growth of the school was slow and steady. Classes of less than 20 were graduated up until the twenties, when classes increased to 25. The income was slender but adequate for the small school, and a spirit of friendliness and cooperation between faculty and students existed that is remarked on by many graduates.

The most critical times came in the thirties, when disaster of major proportions struck the state. The depression and years of drought combined to drop the total state income from \$315,000,000 in 1929 to \$111,000,000 in 1932. More than a third of the farmers lost their farms, and 230,000 people—more than a third of the population—were on relief in 1937. In 1937, more than a third of the taxes were delinquent. The whole University suffered from the drastic cuts in state appropriation, and the salary for deans was cut from \$4,800 to \$1,920. Entrenchment was general. Letters show that medical teachers canceled subscriptions to their scientific journals and withdrew from societies. Still,

the school struggled on with never a break in classes and never without a group of graduates who transferred and did well. In 1936, the school was dropped from membership by the Association of American Medical Colleges. In 1939, even the probationary status of the school was threatened by the American Medical Association.

In the early forties, increased rainfall and a good grain market enabled the state to meet the needs which it had so long recognized but had been unable to fulfill, and the School of Medicine entered its final current phase. In 1945, the state legislature created the North Dakota Medical Center and appropriated \$250,000 for the construction of a medical science building and, in 1948, ratified a mill levy, which, for the first time, provided adequate funds for the maintenance of medical education in the state.

In 1947, Alfred Lawton, M.D., became dean. After serving one year in this position, he left to administer the extensive research programs of the Veterans Administration and was succeeded by Wilbur F. Potter, M.D. A man excellently qualified as a teacher and medical educator, Dr. Potter became dean and professor of physiology in 1948. At the time he came to North Dakota, things were at a low ebb. There were only three full-time men on the faculty, and a building had yet to be built. In the five years of his deanship, Dr. Potter oversaw the construction and equipment of a new medical science building, assembled a competent faculty, reorganized the curriculum, and created a department of biochemistry.

Upon the recommendation of the accrediting agencies, a clinical clerkship was established in 1952, whereby the sophomore students at the end of their second year are assigned to various teaching centers throughout the state where they spend a month gaining experience from bedside teaching. This clerkship has been most rewarding, serving to better prepare our students for transfer.

In 1952, the School of Medicine was fully accredited by the American Medical Association and the Association of American Medical Colleges and took its place as one of the approved two-year medical schools of the nation. Dr. Potter resigned as dean in 1953 in order to devote his full time to teaching. He was succeeded by T. H. Harwood, M.D., previously assistant dean and associate professor in the department of internal medicine at the University of Vermont College of Medicine.

The medical science building, completed and occupied in 1953, is a 4-story, Gothic structure,

with a penthouse on the roof for animal quarters. In addition to the student laboratories, each department has its own research laboratories which provide adequate space for the considerable amount of research carried on each year.

The medical library, appropriately named the Harley E. French Library, has its reading room on the first floor and 5 floors of adjacent stacks. The library has, at present, 7,424 bound volumes of journals, 5,933 books, and 1,273 books in the nursing library. The library subscribes to 251 journals.

The pathology department has a well-stocked, excellently lighted museum, which, with study tables, anatomic models, and other instructional devices, is a teaching area rather than merely a storage space.

Research had been relatively nonexistent until space and laboratory facilities were made available in the new building, and the staff had been increased to a proper functioning level. Since 1950, the amount of research work done has steadily increased in the areas of anatomy, pathology, biochemistry, virology, and physiology, supported by grants from the North Dakota Cancer Society, the United States Public Health Service, the United States Atomic Energy Commission, the American Cyanamid Company, the Bremmer Foundation, the American Medical Association, the United States Air Force, the National Foundation for Infantile Paralysis, and the National Vitamin Foundation. For several years, between \$80,000 and \$100,000 have been available from these sources for research. In 1952, Mrs. Bertha Ireland of Grand Forks gave a sum of money creating the Guy and Bertha Ireland Cancer Research Laboratory, which is under the director of the department of biochemistry. The department maintains an isotope laboratory for the more modern technics involving radioactivity.

A graduate program was inaugurated in 1948, offering graduate work in the departments of anatomy, bacteriology, biochemistry, pathology, and physiology. A doctorate program was set up in the departments of anatomy and biochemistry, and the first doctorate degree was conferred in 1955. The department of pathology has an approved program for residency training.

The School of Medicine expanded its function in 1949 by establishing an approved School of Medical Technology. Its first class graduated in 1953.

The Medical Center, further recognizing its responsibility for training allied medical personnel, authorized the construction of a rehabilitation, outpatient, and treatment diagnostic center

in January 1956. It is situated adjacent to the School of Medicine. This facility strengthens the teaching programs of occupational therapy, physical therapy, clinical psychology, and social services. Trained personnel in all of these areas are badly needed in North Dakota.

In common with most state supported schools, the Medical School at North Dakota gives first preference to residents of the state. Most years, there has been room for 10 per cent of the class to be filled by out-of-state students. Each year our students transfer without difficulty, and, on the whole, do well after transferring. No formal

arrangements exist for transfer, although several schools have been working closely with North Dakota for many years. Students have transferred to a total of 41 schools in the past ten years.

It is gratifying after years of struggle to see the recognition recently given to two-year schools by the accrediting agencies, which see them as valuable in providing students in the clinical years for schools which have bottlenecks in the basic sciences. They have advocated the formation of more two-year schools, vindicating the plans of Dr. Brannon and Dr. French.

LARGE SUBSTERNAL goiters can be removed by the standard cervical approach with low rates of morbidity and mortality.

The most decisive factor in the selection of a cervical approach is the location of the goiter. If the uppermost portion of a substernal goiter is roentgenographically no lower than the jugular notch, a cervical incision will be satisfactory.

Properly conducted, a standard thyroidectomy through a collar incision is safe, simple, and effective. This approach allows complete control of arterial and venous blood supply to the thyroid, accurate identification of the laryngeal nerves, and proper preservation of the parathyroid glands.

Surprisingly large substernal goiters may be removed through the neck. When the patient is given local anesthesia, he may facilitate removal of the mass by coughing. In some cases, morcellation of the mass with aspiration of the contents or piecemeal removal of the central portion of the mass with a placenta extractor allows traction on the capsule.

When tracheal deviation is pronounced or the goiter is extremely large, general anesthesia may be administered by intratracheal tube. However, morbidity is somewhat greater with general than with local anesthesia.

It is rarely necessary to split the sternum. Some unusually large goiters do require thoracotomy, especially when cervical thyroidectomy has been previously performed and the recurrent goiter lies entirely within the chest.

Before 1950, the thoracic approach was used only 17 times in 40,000 thyroidectomies. From 1950 to 1956, the thoracic route was employed 24 times in 5,819 thyroidectomies. Substernal goiters were present in 112 of the latter group. Of the 24 substernal goiters removed through thoracic incisions, 20 had originated in the right lobe. The average length of hospitalization was eleven days. However, for the 88 patients who had cervical thyroidectomies for substernal goiters, the average length of hospital stay was six days.

E. S. JUDD, O. H. BEAIRS, and D. E. BOWES: Consideration of proper surgical approach for substernal goiter. *Surg., Gynec. & Obst.* 110:90-98, 1960.

The Ghost Medical Journals of Minnesota

I. *The Minnesota Homœopath*

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THE FIRST PHYSICIAN to practice in what was to become the state of Minnesota was a "regular" practitioner,¹ as was the physician who made the first contribution to a medical journal from Minnesota.² But the first medical journal in Minnesota was a fugitive little homeopathic sheet of one hundred one years ago, during the midterm of President James Buchanan, when the Federal Union was all but torn asunder.³⁻⁵

This journal was *The Minnesota Homœopath* (figure 1), a virtually unknown publication issued in St. Paul by one Dr. George Hadfield (figure 2) in 1858 and 1859. Scarcely anything more is known about the publisher than is known about his journal. Number 4 of volume 1, owned by the Minnesota Historical Society, is the only known example of this odd piece of ephemera.

Whether or not *The Minnesota Homœopath* was directed exclusively to the attention of the medical profession is conjectural; it may have served an additional purpose in being circulated generally to the public. In 1859, Minnesota had been a member of the Federal Union only a year, and Dr. Hadfield may have been one of the boomers who rushed to the Territory of Minnesota in anticipation of an extensive clientele when the territory should become a state. The Minnesota census of 1857, conducted preparatory to the admission of Minnesota to the Federal Union, showed that Hadfield in that year was 43 years old and that he had been born in England.⁶ An unknown writer in the *Transactions* of the old Minnesota State Homœopathic Institute wrote that "In St. Paul Dr. Geo. T. Hadfield, who came in 1854, was the first practitioner who stayed."⁷ Vedder⁸ wrote in 1890 that "Dr. George Hadfield was a homeopathic physician, and died here [St. Paul] at the beginning of the War of the Rebellion." This statement certainly is open to question, for, in chapter 81 of the *Transactions*,⁹ it is indicated that "Geo. Had-

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The Minnesota Homœopath.

Published bi-monthly, by Dr. George Hadfield.

Vol. 1.

ST. PAUL, JANUARY, 1859.

No. 4.

Homœopathy.

Dear reader, I hope you will excuse me intruding upon your time a few moments while calling your attention to a subject of so much importance, one in which every individual upon the face of the earth is more or less interested,—The art of healing; and as long as the human family are subject to disease they will naturally feel some interest in being cured by the most "prompt, mild, and permanent manner."

It is not necessary for us to go into any detail of the amount of mind that has been engaged in investigating the science of medicine, or the amount of life sacrificed and money expended since the days of Hippocrates to the present moment, nor to say anything of the uncertainty, dissatisfaction, and frequent acknowledgements of the inadequacy of the "healing art," by the most scientific minds; these are too palpable to need any detail from us.

Hahnemann felt this lack of knowledge, this uncertainty of prescribing, this want of law or tangibility which all other sciences had, or were assuming; and being a thorough scholar, and having a benevolent heart, a conscientious spirit and investigating mind, he quit the practice of medicine and devoted his whole time to the investigation of chemistry.

While engaged in translating Cullen's *Materia Medica* into his mother tongue, his attention was arrested by the fact recorded by Cullen, that a person having taken Peruvian bark by mistake, was soon after attacked by rigors similar to fever and ague, for which that bark is so notorious a specific.

Hahnemann could not receive this statement as a freak of nature without further proof of its validity; consequently he took the drug himself and soon found himself in the situation described by Cullen, and after waiting until the drug had spent its strength upon his wiry constitution, and nature had thoroughly rallied, he again and again repeated the trial, and the results were invariably the same. His mind then turned to reflect upon the numerous volumes of medical history and medical practice which he had read, and finding that numerous writers recorded mercury as a specific for syphilis, and also that cases were recorded by as equally observant and scientific men, of mercury having produced a state similar to syphilis, that this accorded with the views of Hippocrates, that diseases were cured by drugs capable of producing similar diseases when administered to persons in perfect health. Hahnemann then set about proving other drugs which had been said

Fig. 1. *The Minnesota Homœopath*, Vol. 1, No. 4, January 1859. From the files of the Minnesota Historical Society

field, Cincinnati, Ohio," had been made an honorary member of the institute in 1869.

Armstrong³ said that Dr. Hadfield had an office on Fourth Street just below Seven Corners in St. Paul, and that in 1860 he went first to Memphis, Tennessee, and then to Little Rock, Arkansas. After the War of the Rebellion, he



Fig. 2. Dr. George Hadfield. From the files of the Minnesota Historical Society

settled in Chicago, and he moved to Cincinnati in 1869. There he "exploited a machine for equalizing the circulation of the blood."³ Apparently, Dr. Hadfield had some part in bringing another homeopathic physician, Dr. Heinrich Alexander Leopold von Wedelstaedt (1817-1900), to St. Paul in 1857.^{3,10}

Two other journals devoted to the homeopathic practice of medicine followed Dr. George Hadfield's curious little *Minnesota Homœopath*. These will be described in sequence.

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NO CHARACTERISTIC abnormality is found upon histologic examination of aortas from patients with dissecting aneurysms. Occasional alterations of structure affecting one or more elements of the media are noted, but similar changes are present in controls. Many patients with dissecting aneurysm show normal histologic appearance of the aortic wall. A disturbance in the metabolism of collagen may be the basic defect underlying aortic dissection, and the closely related connective tissue ground substance may also be involved. When the patient is pregnant or suffering from coarctation of the aorta, the cause of the connective tissue defect appears to be hormonal; in patients with Marfan's syndrome, the defect is hereditary; with experimental lathyrism, the cause is a known toxin; but with the common form, occurring in middle-aged persons with arterial hypertension, the cause of the connective tissue changes responsible for the defect is not known. It has become apparent that hypertension is not solely responsible.

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Frank F. Wesbrook, M.D.

Action in Medical Education and Public Health in Minnesota

S. MARX WHITE, M.D.

Minneapolis

BORN IN Brent County, Ontario, July 12, 1868, Frank F. Wesbrook came to the University of Minnesota at the age of 27, left to assume the presidency of the University of British Columbia at 45, and died in Vancouver, British Columbia, on October 21, 1918, at 50 years of age.

He had graduated from Manitoba Medical College, Winnipeg, in 1890, and then took a summer course at McGill University, Montreal. A year at Rotunda Hospital, Dublin, followed. He won a British Medical Association and a John Lucas Walker fellowship in pathology under Professor Roy at Gonville and Caius College, Cambridge, England. He gained medical experience at King's College and St. Bartholomew's hospitals in London and then spent several months at the Institute of Hygiene and Pathology in Marburg, Germany, where an epidemic of cholera was under study. This fundamental training laid the foundation on which his native research ability was built. He returned to Winnipeg as professor of pathology, but his stay there was very brief, for he came almost at once to the University of Minnesota as professor of pathology and bacteriology in the fall of 1895. In the spring of 1896, he was appointed bacteriologist to the Minnesota State Board of Health. The very limited equipment of the board's laboratories, then housed in the Mechanics Arts Building on the campus, was transferred to the medical school's new building for laboratories, later to be named Wesbrook Hall.

The arrangement of the new laboratories was entrusted to Dr. Orianna McDaniel. Dr. McDaniel had received her guidance and inspiration from Dean Vaughan and Professor Navy and had graduated

from the medical school of the University of Michigan in 1894. She performed a notable service in the Minnesota State Board of Health over a period of fifty years, retiring in 1946.

A paragraph is needed to sketch the milieu into which Dr. Wesbrook was inducted on his arrival. The career of Dr. Charles N. Hewitt has been outlined in Folwell's *History of Minnesota*. Responsible in large part for the establishment of the Minnesota State Board of Health in 1872, he at once became its first secretary. He continued practice in Red Wing, Minnesota, about 40 miles from the capital city, commuting by rail. Much of the work of the board was done in his home town. In 1890, Dr. Hewitt began the manufacture of smallpox vaccine, which he supplied at cost to physicians in Minnesota. In 1894, the very limited equipment of the chemical and bacteriologic laboratories was removed to the campus of the University of Minnesota. Dr. Hewitt's salary at the beginning of his term of service was \$200 per annum. At the end of his term, it was \$1,500. Of distinguished New England parentage and educated in the arts and in medicine, he was a man of extraordinary energy, determination, and courage. He worked tirelessly to abolish the primitive local lines of authority existing and to correlate the state and even national health efforts. He was honored in England and France, and his college conferred on him the degree of Doctor of Laws.

Folwell has written, "Charles Nathaniel Hewitt is hereby written down as Minnesota's apostle of Public Health."

On June 11, 1897, Dr. Hewitt learned that he had not been reappointed to the board. He left abruptly

never to return. Dr. Henry M. Bracken, a member of the board, was appointed in his place. Dr. Hewitt never revealed his understanding of the reasons for his dismissal. David M. Clough had become governor of Minnesota. Jordan has recorded that Dr. Hewitt had been asked to contribute to the Republican campaign fund. He refused, stating that the state board of health had never mixed in politics and never would with his consent. This attitude of Dr. Hewitt's of standing foursquare against any participation in partisan political affairs by public health officials has been continually observed in Minnesota and is a source of the high quality and uninterrupted efficiency characteristic of such affairs here not only in the state board but in the other specialized health fields, such as tuberculosis.

The situation as it concerned the medical school at the time of Dr. Westbrook's arrival can also be briefly told. Three schools—the Minnesota Hospital College, the Minnesota College of Homeopathic Medicine, both in Minneapolis, and the St. Paul Medical College—had agreed to surrender their charters in order that a teaching department of medicine might be established at the state university. This was done, and, in October 1888, entrance examinations were held under the supervision of Dr. Perry Millard, who had become dean. Dr. Hewitt was not included in the reorganization, although he still held his post in the board of health. In the new University of Minnesota Medical School, all students received their instruction in the medical sciences without distinction during the first two years, while, in the last two years, the clinical subjects were taught in separate schools, the regular and the homeopathic. This was a strange arrangement, for there were separate clinical departments, such as surgery and obstetrics, as well as medicine and therapeutics. The last two mentioned, of course, were the only departments truly representative of the cult.

The sources of material for study in Dr. Westbrook's department were at first a jumble. An assistant in the department began visiting operating rooms and clinics in the private hospitals for pathologic material and cultures. Physicians throughout the state occasionally sent in pieces of tissue removed at operation. One very active alumnus from 150 miles away was especially industrious and is recalled to memory for the abundance of material removed at operation and the paucity of information accompanying it. It was as if he challenged the staff by saying, "You tell me what this is!" A Minneapolis surgeon used to send post cards through the mails inviting physicians to visit his weekly clinic in a private hospital, and the writer had the task of visiting these for material. These clinics are remembered best for operations performed in haste and the spattering ability of the surgeon. While I never actually saw blood on the ceiling, I often was reminded of a canary in a birdbath.

Hamline University had established a medical department with its buildings across the street from the Minneapolis General Hospital. The first building

of this hospital was a very large residence in the center of a block between Fifth and Sixth streets and Fifth and Sixth avenues, nearly 2 miles from the state university. Buildings were added gradually. The staff was made up of physicians and surgeons from the Hamline school, the University of Minnesota faculty, and local physicians who were not connected with either institution. The rivalry was insistent and interfered with the development of any kind of strong teaching or research services.

With Dr. Westbrook's help, an autopsy service was established in the old barn attached to the residence serving as the Minneapolis General Hospital. A box stall was walled in, and an autopsy table was built. The autopsy protocols had to be handwritten in a large ledger like a tome, since the pathologist had no secretarial help. Some of the reports are incomplete because of the press of teaching work. It was not long before Dr. Westbrook began plans for a new building on the campus, to be called the Institute of Public Health, which would house both the expanded Department of Pathology and Bacteriology and the laboratories of the state board of health. When this was completed, the building now named Westbrook Hall was left to the Physiology Department, under Dr. Richard Olding Beard, and to the Department of Histology and Embryology, under Dr. Thomas G. Lee.

Dr. Westbrook developed very cordial relations with the legislature, especially with rural members. He planned trips of inspection during the periods of legislative assembly. Through these trips and his cordial and helpful contacts with physicians throughout the state, he was able to impress legislators with the needs of the medical school to the point that this school was suspect with some of the less impressive departments of the university. An account of certain matters concerning legislative appropriations is of interest. Dr. Westbrook made his plans far in advance. Most projects had to be presented to 2 or 3 of the biennial sessions in advance of the session in which the project could be actuated. It was thus an educational process.

In those days, the legislature had a wary eye out for that upstart university in the big city that was beginning to cost so much of Minnesota's hard cash. The happy faculty had developed of cutting the requests by 15 to 20 per cent each time they were presented.

When George Edgar Vincent came from the University of Chicago as president of the University of Minnesota in April 1911, he promptly saw the absurdity of this procedure. When the first request for appropriations was sent to the Capitol after he had time to get a firm grasp on the situation, he very competently and confidently had the matter put in the form it should properly take. All requests were to be completely realistic. Only the absolutely irreducible needs were to be included.

With his experience, Dr. Westbrook favored the educational process and presented his evidence as courteously as he could. He was overruled. The

irreducible needs were presented with fervor, conviction, and confidence. The legislature surely could not cut such a carefully and conscientiously worked out request. Imagine the astonishment when the usual cut of about 15 per cent was made. The next legislative session was presented in the historic educational pattern by a sadder and wiser Mr. Vincent. It is said that, in his earlier years, he once wrote a book on the psychology of the mob. The psychologizing of that effort didn't seem to work on the group he now dealt with. In his dealings with the medical profession and the public as director of laboratories of the state board of health, Dr. Westbrook followed the same pattern of education, persuasion, and helpful cooperation.

When Henry M. Bracken succeeded Charles N. Hewitt in 1897 as secretary of the board, the new regime promptly came into action. Great as was his accomplishment and reputation in the field, it might not be inappropriate to call Bracken "the stormy petrel of public health in Minnesota." With him, there seemed to be no intermediate tones. All was either black or white. If a health officer, a practicing physician, or a citizen stepped over the boundary between rectitude and original public health, Dr. Bracken felt it his duty to inform such a one in no uncertain terms where lay the lines. He pounced with beak and claw. He wrote innumerable letters, and they were masterpieces. He called attention to the statutes and the regulations. His invective was sometimes in words of one syllable. Dr. Westbrook was meticulous in seeing that his laboratory authority did not invade the province of the secretary of the board, but, within these limitations, many letters routed through him failed to be sent or were preceded, accompanied, surrounded, or succeeded by an approach of a very different character. He was a master of tact and diplomacy, and he won understanding, cooperation, and support where sterner dealing would fail.

During the period of his career in the state board of health, Dr. Westbrook's chief technical contributions were in rabies, in typhoid, and in the classification of B-diphtheriae. In the last named field, the classification of the various forms of the organism and of the so-called pseudodiphtheria and diphtheroid forms published in 1900 has become a classic, as it laid a foundation which, though slightly modified in some ways, has never been shaken. This was the cooperative work of Dr. Westbrook, Dr. Louis B. Wilson, and Dr. Orianna McDaniel. Dr. Wilson was appointed director of the pathological laboratories of the Mayo Clinic in 1905 and later became director of the Mayo Foundation in Rochester. Dr. McDaniel later became director of the Division of Preventable Diseases and, in 1907, director of the Pasteur Institute.

Dr. Westbrook and Dr. Wilson initiated in the state board laboratories the studies of Rocky Mountain spotted fever, which was prevalent in the Bitter Root Valley in Montana. Minnesota authority and resources were not sufficient to pursue the study.

This was then undertaken at the University of Chicago by Dr. Howard Taylor Ricketts for whom the Rickettsia group of organisms is named. The laboratories here served as a way station for Dr. Ricketts on his way to and from Chicago and Montana, but the work was now all his.

H. A. Whittaker joined the board of health laboratory forces in 1907 as a chemist, became chief of the water and sewage laboratories in 1909, and professor of public health engineering in 1946. With an international reputation in his field, he has been a consultant with the World Health Organization in Geneva and is now a member of the expert advisory panel on environmental sanitation in Washington, D. C.

GROWTH OF THE MEDICAL SCHOOL

While deeply involved in the multiple university laboratory and health board duties and working with inadequate facilities, Dr. Westbrook planned and worked strenuously to aid in developing the clinical departments and to provide a teaching and research hospital. But, first, the campus must be enlarged and the Great Western Railroad tracks which formed a natural barrier at the southern boundary must be removed. He was the motivating force in arousing the Medical Alumni Association to secure funds for expansion of the medical portion of the campus. A sum of \$42,000 was readily subscribed for this purpose, and a tract of 10 acres was secured just south of the railroad tracks and east of the Mississippi River above the bend where it begins its eastward course. This tract was thus just south of the former medical campus where stands Westbrook Hall and the Institute of Public Health Building.

It at once became apparent that not only the medical department but the whole university was ready for expansion. The general alumni now assumed the task. Thus, the greater campus movement was initiated by the medical alumni.

The site for the medical campus was moved to the area in which we now find Elliott Hospital, Millard Hall, and the Mayo Memorial Building. It was not until shortly after Dr. Westbrook assumed the deanship that the legislature made its appropriation of \$450,000 for campus extension. Dr. Westbrook's relationship to the Mayo brothers became close at an early date. He made frequent visits to Rochester for the purpose of conferring with them regarding medical education and hospitals. He early recognized the influence that these men had with the legislators, especially those from rural areas. He secured the support of the Mayo brothers for development of the medical school. It was my privilege to accompany him to Rochester on several occasions. These visits were cordial and very productive of sympathetic understanding. On one of these, an amusing example of the use of mnemonics occurred. Why my name should be any more difficult to remember than Smith or Jones I do not know, but, for the three days that we were entertained in Dr. Will Mayo's home, I was Dr. Snow in Mrs. Mayo's book.

Dr. Thomas S. Roberts, later director of the Museum of Natural History of the university and author of *Birds of Minnesota*, was at this time actively practicing medicine and teaching pediatrics in the medical school. In his practice, he had an interesting arrangement with a very considerable number of the older and leading families in town. A retainer, which I believe was \$250 a year from each, gave a prior call on his services, and it also made it possible for him to pursue his bird studies in which they were interested. He sent and often took his surgical patients to the Mayos. With Dr. Wesbrook, Dr. Roberts was the leading factor in securing Dr. William Mayo's interest in the medical school and his appointment in 1907 to the Board of Regents. Here he served until his death and contributed in great degree to the development of the medical school. All the world may see and know of the memorial to the Mayo name on the university campus.

The practice of taking patients from this medical center to a group of surgeons 90 miles away in a little country town did not meet with general approval among the surgeons here in those earlier days. In 1913, arrangements were agreed upon by which affiliation with the university was established through the Mayo Foundation for Medical Education and Research. At first temporary, it became permanent on September 17, 1917. Although, naturally, there would have been some opposition to this unique arrangement, the discussion which occurred after Dr. Wesbrook left in 1913, if it occurred at all, would have been much less disagreeable to all concerned had he been present. His tact and skill would surely have guided the affiliation through in ways less acrimonious.

Dr. Wesbrook had been a most—assuredly, the most—significant factor in his time in progressive movements involving the medical school, and they come one upon another in rapid succession.

In 1908, by adoption of the faculty of the medical school of Hamline University into the University of Minnesota Medical School, he abolished the crippling rivalry for clinical material at the Minneapolis General Hospital. At the same time, he persuaded the staff of the homeopathic school in the university that union with the regular staff would be an advantage. Therapeutics and the purely medical subjects were made elective. This was done quietly and with little fanfare. The catalogue of the medical school for one year only has any record. In that, for 1910 and 1911, there is listed an elective by the professor of homeopathic materia medica. After three years, no student chose it. There is no mention of it before or since. The unification of medical education in Minnesota has been a source of greater strength.

To secure a hospital for teaching and research on the campus had been Dr. Wesbrook's objective long before his appointment as dean in 1906. When ground for the greater campus was secured, many fine residences were found in the area. Two very large ones at State Street on the north side of Wash-

ington Avenue were made available. One was assigned to medicine and one to surgery. They were in use for two years.

What if surgical patients had to be carried up or downstairs to or from the operating room? Dr. Amos W. Abbott, professor of gynecology, had had to do this. Until the William H. Dunwoody bequest initiated the greater Abbott Hospital, his work was carried on in a building at 10 East Seventeenth Street converted from a small apartment building without elevators. Resources in the university permitted only part-time employment for staff members in the clinical departments. Dr. James E. Moore was given charge of the surgical services and Dr. Charles Lyman Greene of St. Paul of the medical. These headships were projected into the hospital soon to be built.

By 1910, 3 buildings had been equipped on the new site. The Medical Science Building, later to be named Millard Hall, and the Institute of Anatomy had been completed at a joint cost of \$636,000. Elliott Memorial Hospital with 120 beds was the gift of the late Dr. A. F. and Mrs. Elliott and the administrator of their estate, Mr. Walter J. Trask. To this benefaction amounting to \$120,000, \$88,000 was added by appropriation for completion and equipment. Dr. A. B. Cates, professor of obstetrics, was instigator in securing this, the first of many gifts for hospital construction.

Shortly after this time, the removal of the Great Western Railroad tracks was accomplished. They ran through a deep cut now covered by the Northrop Memorial Stadium and Auditorium. Thus, the greater campus was unified.

The project foremost in Dr. Wesbrook's thoughts and plans was a medical school in which public health should be integral with clinical medicine in the training of the physician. Dr. Wesbrook, as has been indicated previously, appreciated profoundly the crucial part the physician plays in public as well as in private health matters. He wished to indoctrinate more insistently than ever before while the neophyte was studying the fundamentals. His writing during the period 1906 to 1913 reflects this urge.

The impress of William Watts Folwell and especially of Cyrus Northrop will remain with us. During his relatively short term, George Edgar Vincent put the institution on the way toward being a great university. Not too long after his arrival, the medical school caught his eye. It admittedly was growing more rapidly than any other segment, and its progress was widely heralded.

Dr. J. N. McCormack was executive officer of the Kentucky State Board of Health from 1883 to 1913. As traveling organizer of the American Medical Association, he had visited all but a very few counties in the United States and was familiar with the character of medical practice throughout the nation. On several occasions from about 1908 to 1912 at various medical meetings in places as far from Minnesota as San Francisco and New Orleans, the writer heard him state that the general level of medical

practice in Minnesota was the best in the nation. This, of course, did not mean that Minnesota's medical school was the best. Such schools as Harvard, Johns Hopkins, and the University of Michigan, and schools in Philadelphia, New York, and Chicago were much better organized, and many practitioners came here from medical schools other than Minnesota's. W. J. Mayo came from the University of Michigan, and C. H. Mayo came from Northwestern in Chicago. Nevertheless, Minnesota's school was playing a significant part in the character of medical practice in the state.

Dr. Wesbrook realized that the time had come when the clinical departments required men who could give their whole time to teaching and research. He planned to accomplish this as rapidly as proper hospital facilities could be provided. Many of the local men had devoted much time and effort to the school without compensation. As rapidly as possible without failing to recognize their services, they were to be replaced by full-time men. Mr. Vincent's plans were in accord, but he had no background of knowledge of the character or extent of these services and desired that progress be as rapid as possible. Dr. Wesbrook had many sessions with the president and, at one time, had come to a definite agreement that reorganization would be fostered as rapidly as could be done but no specific announcement would be made. Within a few days after this definite agreement had been made, the president spoke at a meeting in St. Paul. Evidently his enthusiasm mounted with his well-known rapid-fire diction. His memory slipped momentarily, for he announced that he was about to reorganize the medical school. This, of course, came as a severe shock in many quarters. Much talk gave Dr. Wesbrook great distress. To him, it was a severe blow to have the onset so flamboyant. This he was trying sedulously to avoid. He planned a healthy evolution, not revolution. Dr. Mayo would have called the sudden move "a balloon ascension." In 1921, eight years after Dr. Wesbrook's departure, a survey committee of three of the most eminent medical educators in the country reported to the Board of Regents that "There is no reason why the heads of clinical branches should at present be made full-time men." The committee consisted of Dr. Frank Billings of the University of Chicago, Dr. J. M. T. Finney of Johns Hopkins University School of Medicine, and Dr. Victor Vaughan of the University of Michigan. Though not known to be in the very forefront in teaching and research, the medical school had a history of a school in which the highest ideals of medicine were inculcated. Clinical experience was freely recorded by the staff.

Before Dr. Wesbrook's brief period, no support and no opportunity for whole-time clinical services existed. There were no hospital facilities in which the type of research later developed here could be fostered. Even laboratory facilities were extremely limited. He was caught in the web into which many able research men have been forced. Trained for research and fulfilling this destiny early in his career,

it was his fate to expend his compelling energies in building in order that opportunity might be provided for others. His tenure was too brief for him to see the results of his constructive efforts. As Mr. Vincent had set the university in its modern way, so had Dr. Wesbrook wrought and built to set the medical school. Elias P. Lyon was privileged to build greatly on foundations that Dr. Wesbrook had laid. When he left here in the spring of 1913, he did so to become the first president of the new University of British Columbia. Here a full appreciation of the significance of his plans was entertained, and here he was to be given a free hand in the development of a medical school and a school of public health in close cooperation with the provincial department of health.

REASONS FOR LEAVING THE UNIVERSITY

Why did he leave a project he was bringing so well on its way for one so embryonic? The reasons were both complicated and simple. I can speak with some authority, for I was closely in his confidence, having spent the first ten years up to 1908 in his department and then joining the department of medicine where clinical opportunities were being developed. To return to his native Canada and to a young university where he could carry out his plans unopposed would naturally be a great opportunity.

President Vincent had recognized at an early moment in his tenure that Dr. Wesbrook was a man of great power with a strong following in the community. At the time, the medical school was making greater progress than any other division in the university. Mr. Vincent's ideas about public health and the medical school did not follow Dr. Wesbrook's, and the latter was deeply concerned. This is not a medical treatise but such factors entered into Dr. Wesbrook's consideration.

I had found in 1910 that he had arterial hypertension. It seemed mild and was extraordinarily labile. There were then no effective antihypertensive drugs; the *affekt* was the only effective approach in management. Highest readings recorded were 220 systolic and usually 112 or so diastolic but once or twice 120 diastolic, all of these taken when recumbent. When I darkened the room, asked him to relax as if to take a nap, and left him alone in the room but with the pressure cuff on, he would be assured that he would not be forgotten and that on my return and after completion of the study he would be told the exact figures. Ten minutes later, if the examiner spoke in a low toned monologue on pleasant subjects, a remarkable reduction in pressure was often secured. Figures as low as 150 systolic and 90 to 92 diastolic were repeatedly recorded. Thus, a reduction of approximately 70 systolic and 30 diastolic was repeatedly obtained. Now, bring up the subject of the medical school, Vincent, the Board of Regents, or prospects of his plans here for the medical school and public health and at once the previous high reading appeared.

With the prospect of sympathetic support, Dr.

Wesbrook believed that the chances for his continued health and well-being were better in the University of British Columbia. The necessity to struggle against opposition from above prevented any opportunity for uninterrupted serenity or equanimity. The appeal was irresistible. He had been compelled by circumstances to build when he had trained for and hoped to develop his capacities in research. The new opportunity was to make real his vision of a great university medical school and public health program in conjunction and unopposed.

Dr. E. L. Tuohy, who was a student of Dr. Wesbrook and later his colleague, wrote in 1923: "I consider that Wesbrook was responsible more than any other man living or dead for medical undergraduate teaching in Minnesota. The ideals and schedules of teaching, the correlation of all forces for the control and alleviation of disease were all part of his superb consciousness. He blueprinted the prospective development of our Medical School. The influence of this great character and personality became apparent early. The intervening years only attested

Wesbrook's great capacity to stimulate others."

In an address at Manitoba University in 1913, Dr. Wesbrook said, "The people's university must meet all the needs of all the people. We must therefore proceed with care to the erection of those workshops where we may design and fashion the tools needed in the building of a nation and from which we can survey and lay out paths of enlightenment, tunnel the mountains of ignorance and bridge the chasms of incompetence."

Dr. Wesbrook's plans for the University of British Columbia were halted by war almost before they were completed. Not for ten years, that is, until 1923, was active construction begun. H. T. Logan has written that the plans and drawings then submitted have guided in no small measure all subsequent development on the university site.

Dr. Wesbrook died in October 1918, three weeks before the armistice, blinded by a hypertensive retinitis but still full of courage and of his plans for a university with a medical school and school of public health after his own heart.

DIFFUSE OBSTRUCTION emphysema may be diagnosed by a specific physical sign, a roentgenographic sign, and measurement of desaturation time. In patients without emphysema, the inferior right lung border, as determined by percussion in the right midclavicular line, is at or above the sixth interspace. In those with emphysema, it is below the sixth interspace. Percussion is done with the patient in a recumbent or semirecumbent position.

On roentgenograms made during normal breathing, the right diaphragm is likely to be at or below the eleventh rib if emphysema is present. If the right diaphragm is above this level, emphysema cannot be excluded.

In individuals without emphysema, the 90 per cent desaturation time recorded by an oximeter is less than 350 seconds but almost always exceeds this time in patients with emphysema.

C. R. WOOLF: Diagnosis of emphysema: physical sign, roentgenographic sign, and oximeter test; *Am. Rev. Resp. Dis.* 80:705-715, 1959.

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Book Reviews

Leukemia

WILLIAM DAMESHEK, M.D., and FREDERICK R. GUNZ, M.D., PH.D., 1958. *New York: Grune & Stratton.* 420 pages. *Illustrated.* \$15.75.

Authors Dameshek and Gunz have done a superb job. In their preface they say essentially that they are presenting a fairly comprehensive picture of the present state of knowledge of human leukemia, realizing that it is but an interim report. This is precisely what the authors do throughout the book in a very readable and exciting fashion. Every physician will enjoy reading this book, and it is a must for all those who deal with patients with leukemia, be they general practitioners or specialists.

The book is divided into 16 chapters, beginning with leukemia in the past and ending with leukemia in the future. In between is all that is now known about the disease. Not everyone will agree with the authors' definition and classifications, but certainly they present all of the various classifications in a fair manner, that is, in such a way that, whatever the terminology one uses for various leukemic and related states, it can be identified with the authors' terminology, which actually is quite common and very sensible. The discussion on the prevalence and etiology of leukemia summarizes all the basic facts and suggests new avenues of approach toward solving these problems.

The chapter on the pathology of the leukemia cells is excellent, and the illustrations are quite good, as they are throughout the entire book.

The chapters on the course and special pathology as related to symptomatology, the clinical descriptive features, and the special features of leukemia are excellent descriptions of the diseases and its complications.

The chapters on differential and laboratory diagnosis are clear and leave little to be desired. They are followed by a chapter relating leukemia and the myeloproliferative disorders, which puts a good perspective on the interrelationships of these conditions.

There are excellent chapters dealing with prognosis generally and in particular cases and an especially good chapter for the clinician on what one should tell the patient. The improved life expectancy of leukemic patients, which has been attainable during the past decade, is well related to the advancements in therapy during that time. The chapter on treatment is very complete in its scope, dealing first with the mode of action of all the therapeutic agents currently used and secondly with precise directions for the current management of the different forms of leukemia. This portion will be invaluable to anyone who undertakes the treatment of this disease.

Another word should be said complimenting the authors on the excellent bibliographies in each chapter. These appear to include all of the important articles of the past twenty years. The index is very useful, although short. In summary, I feel that this book is a classic work that should be the companion of any physician interested in human leukemia.

DONALD L. NOLLET, M.D.
Hibbing, Minnesota

The Essentials of Roentgen Interpretation

LESTER W. PAUL, M.D., and JOHN H. JUHL, M.D., 1959. *New York: Paul B. Hoeber, Inc.* 815 pages. *Illustrated.* \$25.00.

In the preface, the authors state that their aim has been to set down as concisely as possible what they consider to be the basic facts of roentgen interpretation and to bridge the gap between the elementary text and the multiple volume reference work. This book is designed to be both a review source for practicing physicians, surgeons, and those taking postgraduate training in a specialty and a textbook for the undergraduate medical student. The introduction contains an adequate section explaining the nature of roentgenography and its properties, with brief descriptions of various forms of equipment and their methods of use. The biologic effects of radiation and its hazards are discussed here in a short paragraph, but the authors have included an appendix containing a summary of the current concepts concerning radiation hazards in diagnostic roentgenology.

The text is divided into 6 sections, according to the major anatomic systems. Roentgen anatomy is discussed briefly in each section and subsection. The emphasis throughout is on roentgen diagnosis. Clinical and pathologic descriptions are limited to the information necessary for clarification of the roentgen changes observed. All of the common and a large number of the less common diseases producing roentgen changes are included. Discussion of differential roentgen diagnosis is of necessity limited in a volume of this size and is confined to the commoner conditions.

Methods of roentgen examination are described, particularly those of the more complex diagnostic procedures. The latter descriptions and those of the less common disease entities are set in smaller type.

References have been selected to direct the reader to a wide range of literature. Extensive bibliographies can be obtained from these reference works.

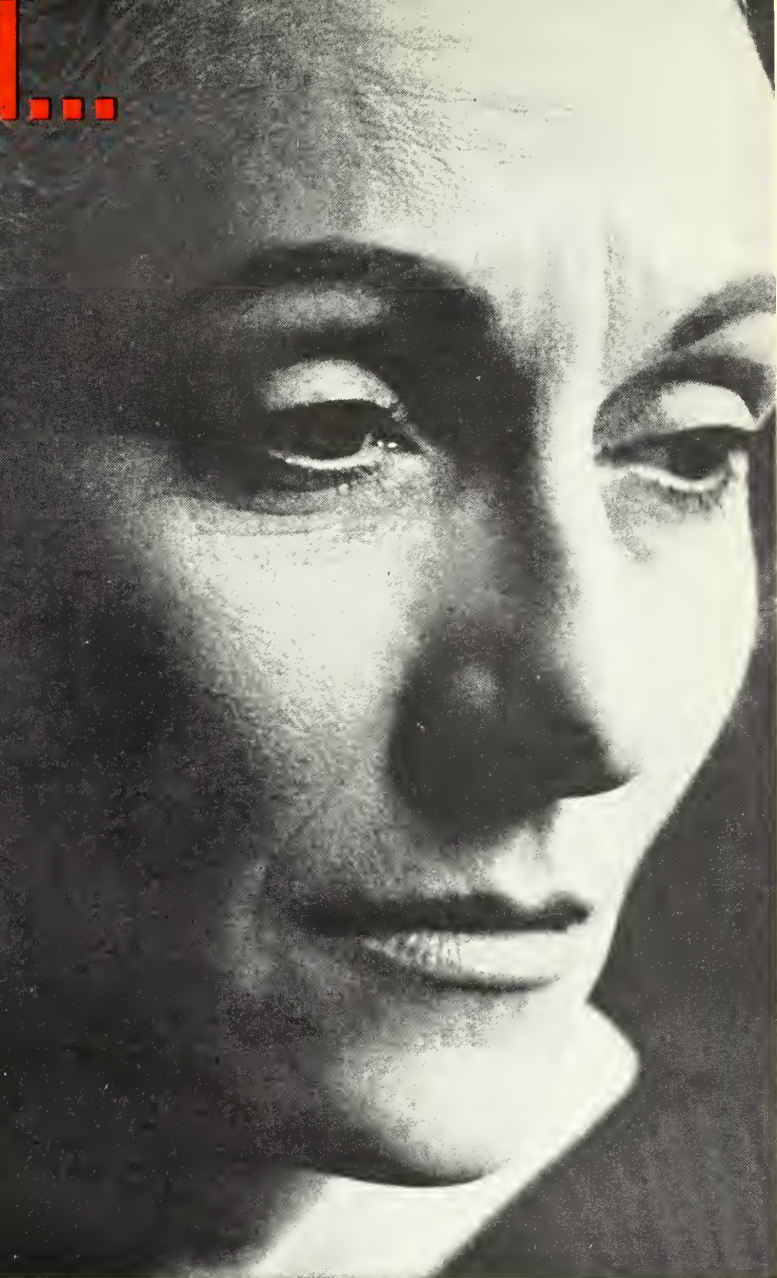
In the text, there is little with which one could quarrel. The language is clear and comprehensive. On most points where doubt or controversy presently exists, the authors have simply stated this without discussion. The many reproductions of roentgenograms are in negative or facsimile form, and the standard of reproduction is uniformly good. Descriptions of roentgen findings are clear and should readily be understood by those not engaged in roentgenologic practice.

Minor criticisms can be made, many of which are due to the brevity forced upon the authors by space limitation. Illustrations of roentgen anatomy could be improved and could be increased in number without adding significantly to the length of the book. Discussion of differential diagnosis could be amplified with advantage, even if the problem of space required that these be set in small type. In view of the readers for which this book is designed, it would have been preferable to insert the section on radiation hazards in a more prominent position, namely, in the introduction. More stress could have been laid on the few radiographic examinations that account for the large bulk of gonadal irradiation in males and females.

(Continued on page 28A)

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References: 1. Bunim, J. J., et al.: *Arthritis & Rheumatism* 1:313, 1958. 2. Silverman, H. I., and Urdang, A.: *Am. Prof. Pharm.* 25:531, 1959. 3. Rudolph, J. A., and Rudolph, B. M.: *Ann. Allergy* 17:710, 1959. 4. Spies, T. D., et al.: *South. M. J.* 51:1066, 1958. 5. Galli, T., and Mannetti, C.: *Minerva med.* 50:949, 1959. 6. Segal, M. S., et al.: *Ann. Allergy* 17:413, 1959. 7. Duvenci, J., et al.: *Ann. Allergy* 17:695, 1959.

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BOOK REVIEWS

(Continued from page 26A)

A number of statements found in different sections of the text would be regarded as inaccurate by most authorities, and there are occasional notable omissions. However, these criticisms are not of major importance, and the authors have succeeded in providing a well-balanced review of diagnostic roentgenology which should be of great value to the undergraduate medical student and those practicing physicians and surgeons who desire a review source in a book of convenient size.

PAUL F. NEW, M.B., F.F.R.
Boston

Cutaneous Manifestations of the Malignant Lymphomas

SAMUEL M. BLUEFARB, M.D., 1959. Springfield, Ill.: Charles C Thomas. 522 pages. Illustrated. \$15.50.

The title of this enlightening volume on diseases of the reticular endothelial system does not do justice to its actual content. Using the skin as a reflector, the author has woven a complete treatise on the malignant lymphomas revealing not only their early and late cutaneous manifestations but also their involvement of other areas. He has taken meticulous care to arrange a systematic study of all phases of these diseases, delving into historical medical records to complete the discussion. Numerous well-selected illustrations in black and white serve as visual aids for the descriptive text. The inclusion of varied opinions of other authoritative investigators, the review of up-to-date experimental observations, the exhaustive lists of references, and the critical discussion of modern therapy render this a good source book, not only for the dermatologist but also for those in other fields of medicine.

The major four topics dealt with are mycosis fungoides, Hodgkin's disease, lymphosarcoma, and immunologic status of the malignant lymphomas. The section on mycosis fungoides is classic and, perhaps, the most inclusive account written to date. Dr. Bluefarb considers this disputed and mysterious condition a distinct entity apart from the other lymphomas. He clarifies the existing confusion which he states has arisen from the improper classification of the types of this disease. According to him, only the classical type of mycosis fungoides is its true form. The "d'emblee" form is nearly always lymphosarcoma, except in very rare cases in which it is the primary lesion of Hodgkin's disease. The "erythroderma" form is a manifestation of either lymphocytic leukemia, Hodgkin's disease, or lymphosarcoma.

The part on immunologic status of malignant lymphomas offers additional study of these conditions. Herein, the author presents a summary of experimental evidence of the disturbance of antibody production produced by the diseases of the reticular endothelial system.

The underlying theme of the book is a separation of the various lymphomas into distinct but related entities. The author writes that, even though an etiologic relationship may eventually be proved, it appears to be incorrect and misleading at the present time, to classify the group as "lymphoblastomas" inasmuch as each presents a distinctive clinical and histologic picture.

A vote of thanks is due Dr. Bluefarb. In one attractive volume, he has included valuable facts which have taken him fifteen years of diligent research to compile. This book is truly a gift to the physicians.

MILTON GINSBERG, M.D.
Baltimore

The Journal Lancet

SERVING THE MEDICAL PROFESSION OF MINNESOTA,
NORTH DAKOTA, SOUTH DAKOTA, AND MONTANA

The Role of the Voluntary Hospital in the Training of Specialists of Tomorrow

OWEN H. WANGENSTEEN, M.D.

Minneapolis

IT IS AN HONOR which I value to have the privilege of being a lecturer in the Jack Friedman series in this magnificent hospital. It was my pleasure to have known the late Dr. Jack Friedman well and to have rubbed elbows with him in our interphase activities at the University Hospital. Large of stature, but larger still in the telling characters which make a fine and strong man—of such sterling stuff was Jack Friedman. In him were intermingled those gentle qualities of generosity, kindness, modesty, and sympathy which make for warm friendships. One need have known him but a short while to know that these attributes of the man were complemented with a keen intellect, great patience, and a spirit of perseverance and zeal that stamped Jack Friedman as a man who stood out above the crowd. Tragic it was that his life was snuffed out by cancer when he had only begun to find his stride in his life's work. He has, nevertheless, left an enduring impress upon those of us who were privileged to have been close to him as well as upon his field of endeavor. On an occasion such as this, it is fitting that we commit ourselves momentarily to the piquet of memory, recalling

OWEN H. WANGENSTEEN is professor and chief of the Department of Surgery at the University of Minnesota.

Paper is the Jack Friedman Lecture presented at Mt. Sinai Hospital, Minneapolis, January 14, 1960.

with gratitude that his life in some manner touched our own. We need his example far more than he needs our praise.

EARLY PROFESSIONAL CONTACTS WITH THE JEWISH COMMUNITY

It is good to be once more with the group that brought this fine institution into being. At one time, as some of you will know, I was rather closely identified with a certain facet of the beginnings of the Mt. Sinai Hospital effort. To be certain, I had nothing to do with the difficult task of assembling money for the purpose. That was the function and care of persons with keener minds and talents than mine.

Let me first tell you something of the origins of my contact professionally with the Jewish people of this community. A few years after the direction of the Department of Surgery fell into my hands at the University, there came to see me one morning a group of six or seven large men, who supported and carried an obviously ailing, emaciated man in the terminal phase of a lingering illness. This contingent descended upon me without warning and filled the small office space (East 201) occupied by my secretary. It was an occasional audience like this that necessitated placement of the "bubble glass expansion" on the entry, which some of you have seen. The group brought a letter from Dr. F. W.

Wittich which carried the information that the patient had been under treatment in another hospital in the state for seven months, undergoing intensive irradiation and treatment with potassium iodide for the management of an aggressive cervicofacial actinomycosis (lumpy jaw), involving the left side of the face and neck. The referral also carried the information that hope for his recovery had been abandoned—in fact, he had been sent home to await the inevitable. Some of you of my generation already may have identified the patient from this meager description. His friends and possibly some of his relatives, who may be here tonight, will entertain no misgivings, I am very certain, over my mentioning his name. It was Louis Heck.

More than a decade earlier, I too had witnessed at the University Hospital a very similar situation, which terminated fatally because of the reliance of the surgical staff upon potassium iodide and irradiation in the management of actinomycosis, which constituted the conventional treatment of that day.

The Emperor's Clothes story of Hans Christian Andersen still hovers too intimately over many a clinical syndrome, puzzling all of us. As a boy on the farm I had made heroic efforts in a couple cows with lumpy jaw to dose them with potassium iodide, a prescription I had found in some veterinary literature on my Father's modest bookshelf. Though it is almost fifty years ago, I can see now that eager, energetic boy persuading an unwilling cow to drain the content of a large bottle. I wonder how many farm boys, like myself, intent only on bringing help to a cow, horse, or pig, subsequently found themselves on the road to medicine. By 1926, I had come to tell patients who had been sent to the University Hospital that my experience with lumpy jaw on the farm had taught me that those cows ended up in South St. Paul. Our patients from the country had no difficulty seeing the analogy implied in the suggestion that it was better to treat the lesion as though it was cancer. Presently I came to appreciate that actinomycosis in man was essentially an anaerobic infection and that removal of the dead tissue by enucleation was as good as complete excision, even though the procedure had to be repeated once or twice. Fortunately, by the time Louis Heck came to see me, that lesson had been learned, and two curettements cured him. I doubt that he could have withstood more. Such were the beginnings of my surgical contacts with this Jewish community. Those of you who knew Louis Heck will appreciate, I

know, what a warm friend he could be. Whenever a close acquaintance of Louis' was ill and was not doing well, if he needed surgery, somehow or other Louis saw to it that the patient's physician did not entirely overlook the University Hospital. The late William A. O'Brien, whom many of you knew very well, once said to me: "Owen, I am starting a campaign to put an N before the G in your name." I replied: "Very nice, Obie, but when you do this, why not take the I out too?"

BEGINNINGS OF MY INTEREST IN THE MT. SINAI HOSPITAL

Let us get back to the facet of the origins of Mt. Sinai Hospital with which I came to be identified. Years went by. Louis had left this temporal scene with a heart attack; at some time in the intervening years I had removed his gallbladder. In fact, my trademark had come to be imprinted on many an abdomen in the Jewish segment of this community during the years following my initial meeting with Louis Heck.

Shortly after the war, I became a neighbor of the Archangel of your hospital. I had not had the privilege of meeting the occupant of that large White House, but one day perched on a stool in the outpatient clinic, I found my neighbor. After having explored a region of his anatomy, though the wound healed very kindly, my newly won friend exuded from every pore a quality of warm friendship, which I was happy to reciprocate. About that time, a number of my Jewish friends had come to think well enough of me to adopt me into an informal group, which they called the Stitch in Nine Club. We usually got together for an annual dinner. On one of those occasions, just prior to the holiday season, we met again and talked over the years' events, as usual. This time, however, there was a new ingredient in the conversation—the need for a hospital for this Jewish community. I interjected: "If you are building a hospital, why not on the campus of the University, or at least near it?"

Those of you who have been close to the project know its history, though the story in its entirety probably never has and may never be told. Warned by the reception which my proposal received that evening in the Stitch in Nine Club of putting the contemplated Jewish Hospital on or near the campus, before returning home I went to acquaint Dean Diehl with the proposal. The hour was late—approaching midnight. It was snowing heavily. The lights in the Dean's house were out, but I was on an important mission. Undaunted and warmed by the enthusiasm which our discussion had engendered, I rang

the doorbell a few times before I was aware of lights upstairs. After an appropriate interval—deans, like kings, sleep lightly, you know—the Dean came down, clad in pajamas and a light robe. He was frankly a bit surprised to see me but admitted me to his living room, which many of you, I am certain, also came to know well. It was probably not a very proper occasion to find one's enthusiasm for so nebulous a project matched by the Dean under the circumstances. I am certain Harold Diehl, great Dean that he was, thought it was information that could as well have been imparted the next morning.

Over the ensuing months, interest in the possibility of bringing Mt. Sinai Hospital near or onto the campus quickened and sharpened up considerably in the faculty of the Medical School. Ray Amberg, wisest of the directors of the University Hospital, all of whom I have known, supported the project enthusiastically. I wish time permitted to give a blow by blow account of the proceedings from their beginnings until the decision was reached by the Board of Regents of the University in December 1949 not to grant the request. However, there is one facet of the story, which I feel I must tell. A committee of the Regents had declined the proposal in November 1949. Your Archangel, flanked by his attorneys, was present at that hearing. There is a quality present in many Norwegians which some of you must have encountered in this community. We of that origin call it persistence, but our friends from outside the Scandinavian peninsula, I note, usually refer to it as stubbornness.

I then said to your Archangel who had negotiated the contract for the building of the hospital on its present site, "What about another go on this important matter with the Regents?" After a few moments' deliberation, he replied, "Give me three hours to think about it." At this time the steam shovels were already at work at Twenty-second and Chicago Avenue. I was back in three hours and the Guardian Angel of your fortunes said: "If you feel a review of the matter before the entire Board of Regents has any promise of a better outcome, I will go along with the proposal. It will probably cost me \$50,000 to fill in the hole in event of approval, but I shall do it." How often in life does one encounter a man of that spirit? Every worthwhile enterprise should have many such friends, but let us all be grateful for and appreciative of one *Jay Phillips!*

Armed with Jay's continued support of the proposal and with President Morrill's approval, I went to the chairman of the Board of Regents of the University and got permission to present

the proposal before the whole Board. Mr. Amberg, hospital director, and Harold Diehl and I pleaded the case of placement of Mt. Sinai Hospital on the campus. Your group had obtained an option on the Motley School site, but that location had been vetoed by the local citizens of that area, and especially by the two local aldermen, persons to whom we normally turn for help in the support of worthy causes. I was much heartened by the reception that the suggestion got at the hands of the Regents, but when an immediate verdict was not forthcoming, I realized that the news probably would not be music to our ears. During the intervening years, many a Regent has said to me: "If only we could have had our way, you would have had your way." Would the Regents of today have looked at the matter differently? I think so. Your officers had given the University assurances relative to the power of appointment, which, to my way of thinking, is *the important item* in all contractual arrangements between institutions. The proposal also had the warm and unanimous endorsement of the Administrative Committee of the Medical School.

It is perhaps not out of place to say here that the strong representations made by Dean Diehl, Mr. Amberg, and myself to the Regents, even though the proposal failed, constituted the entering wedge which drove home to all the officials of the University, the Regents, and our Legislators the pressing need of the Medical School of that day (1948-1949) for expanded hospital facilities. Dr. Morrill, one of the great amongst the succession of presidents of this University, told me on the conclusion of our negotiations that had there been more time, a solution more satisfying to your hospital and the University could undoubtedly have been worked out.

ROLE OF LOYAL WORKERS IN YOUR SUCCESS

There are, therefore, good reasons for strong ties between your institution and the Medical School. Your hospital has been functionalized by a strong staff and by a supporting cast of workers whose devotion and dedication has not been matched in this community and perhaps is unexcelled anywhere in our country. There is only one way properly to acknowledge this kind of gratitude—on one's knees!

However, while we acknowledge with pride and warm admiration the importance of the contributions of a large number of responsible people for the great success of your effort, I wish particularly to commend the loyalty of your wonderful women, who have given so much of

themselves in this endeavor. In the final analysis, the only real gift in life is a part of oneself.

The Good Book tells us that the Creator brought Eve into the world as an afterthought. Yes, it was well toward the end of the second chapter of Genesis that attention was first lent Adam's need for a helpmate. Yes, all animal life on the earth and in the oceans had been brought into being to please Adam before that wonderful second thought brought Eve to his side. Much of man's happiness goes back to this afterthought and, of course, some of his troubles too!

The devoted support which your effort has had from a fine group of dedicated women who participate very actively in your hospital enterprise merits more than a passing word of praise. In fact, I think it would be very much in order to recognize the importance of their contribution by signaling it in an adequate manner—by rededicating yourselves to achievement of desired goals not yet fully reached.

GOALS AND RESPONSIBILITIES

Let us examine some of the needs of your hospital. Success in any endeavor, each of us comes to know, is an enterprise of a lifetime. It is a heavy and recurring responsibility.

What of voluntary hospitals and their responsibilities? In the final analysis, of course, every public institution is a voluntary one; otherwise, it ceases to exist. *Service, teaching, and research* are not responsibilities which relate only to municipal and state hospitals and medical schools. They are functions also of voluntary hospitals—especially so if such hospitals today affect any pretense of standing out in the community or of making an important impress upon the national scene in medicine.

This is an era of change. Nothing stands still. Our enemies of yesterday are today our friends. Witness what has happened with Japan and Germany. After Pearl Harbor, how little did we think that we would or could ever extend a hand of warm welcome to Japanese or Germans. One of our stalwart friends of the last war has since been regarded with suspicion by us. Such are the tides of fortune and life. If we are to ride effectively and smoothly over the obstacles of life, we must catch the wave at its crest and surf in with it. To become embittered by the trials of yesterday sets the stage for tomorrow's defeat.

Any hospital which professes to rise above mediocrity must set goals which are a few cents higher than in other institutions with which it is competing in the community. On my first visit to Dr. William J. Mayo's office, I was impressed

to find on his desk the famous mousetrap quotation from Emerson: "If a man can write a better book, preach a better sermon, or make a better mousetrap than his neighbor, though he builds his house in the woods, the world will make a beaten path to his door." It is still this way with the world. All modern-day hospitals are expected by the public today to render a superior brand of *service*, which reflects warmth and sympathy. We need to imitate the Good Samaritan in our attack upon the problem of community illness. But the hospital that is to stand out must do much more. It must build itself a responsible and distinguished staff. It must continue to maintain a keen interest in research. Your own wonderful laboratory has come to be the prototype for all voluntary hospitals in this area that profess an interest in keeping up with advances in medicine. You have an affiliation with a University which has risen to peaks of greatness here and there in our day. You have distinguished leaders who are full-time representatives of the University at your hospital. They and their activities need support. In accepting affiliation with the University, you have pledged yourselves to the support of a strong *teaching activity* within the hospital. These full-time men who are responsible for the supervision of clinical services and who represent you and the University must have support. They must have beds upon which effective post-graduate teaching can continue. I know your situation in surgery best, of course, but I know you are building strength in other departments too. The present occupant of your full-time position in surgery is strong and competent, as were his predecessors. It is not enough to say that provision has been made for *available* beds. Means and techniques must be devised to see that those beds under the direction of your full-time supervisors *are filled*. How otherwise can these men attract aspirants ambitious for careers in specialties in which your staff is strong? It is through such men, motivated by a desire to become competent specialists, who cherish the hope of contributing through research to the patrimony of knowledge in their chosen fields, that fruition of your own aspirations for this hospital will be accelerated.

At every level of interest throughout your hospital activity, the theme of adequate support of the teaching functions of your directors of medicine and surgery needs to be aired. As a pledge to your dedicated women, who have contributed so importantly to the success of your enterprise, I suggest that your staff and boards alike readdress themselves seriously to the task of filling

these beds. If this is not done, the vision which many of you have had for this hospital cannot fructify. A real effort by all concerned with the fortunes of this hospital needs to be made now without delay toward meeting this important goal.

What is the role of the University in such an enterprise? Its function primarily is to establish desirable goals. Meeting those standards and maintaining them is your responsibility. The University wants nothing for itself. It only wants to help you in creating the kind of hospital development here which will reflect great credit on your activity. Expansion of participation of voluntary hospitals in the training of specialists throughout our country is contingent in large measure upon hospitals, like yours, in metropolitan medical school areas providing and supporting an adequate number of beds to energize the activity of your full-time medical school clinical supervisors. With the wholehearted support of your staff and generous boards, the time may not be far off when, in these areas of interest, the keenest minds of the University may be your own medical scientists, who will add distinction and stature not alone to the hospital but to the University as well.

When a small group of the faculty of the University of Minnesota met with a group of physicians and surgeons in our area seventy-five years ago to discuss the formation of a University Medical School, a member of the faculty suggested that it would be proper to start the meeting with prayer. Let me suggest that you join me in the prayer that the boards of this hospital and its staff will not desist in their joint responsibility of providing and maintaining the teaching facilities which you need to assure realization of the dreams that motivated your founders in bringing this hospital into being. There are a thousand roads to failure but, lamentably, so few to success. You will find that way, I am very certain.

The acceptance of some type of hospital insurance program by a large percentile of families throughout our country has almost eliminated and certainly reduced very materially the number of indigent patients on our hospital wards. They have been supplanted by the part-pay insurance patient who pays all or part of his way under the hospital insurance program. Therefore, your staff, too, must, of necessity, if it welcomes the opportunity of working in this great institution, make its contribution to the pressing necessity of seeing to it that the beds for the full-time clinical directors are occupied. When this important facet of your problem was

discussed with members of your board not so long ago, one of your staff made the suggestion that, if each member of the staff would send two patients a year to each of the clinical services of your full-time University representatives, the committed number of beds (10) on each clinical service could easily be occupied all the time. This is, I believe, a very fair appraisal and is certainly a manner in which that objective can be achieved. A monthly or quarterly rotation of names within your staff will assure continuance of this important facet of your program. Such a device, I believe, will set the stage for automatic perpetuation of your plan. This item is so important that it cannot be put off if your enterprise is to thrive. Moreover, I am told that in other voluntary hospitals in this area, the theme which I have just been developing has gotten off to a nice start with staff cooperation. This hospital and its staff, I feel very certain, is not to be outdone on this score by other voluntary hospitals in this area.

The hospital of the future may very well come to reflect the attitudes and objectives of present day group clinics. Perhaps not so many years away, you may come to be known as the Mt. Sinai Hospital Clinic. Would such a group be slow to assure support of the activity under discussion? I shall leave that answer to you.

Many years ago, I operated upon a patient, let us call her Mrs. A., for a malignancy. The patient was very large and had refused elsewhere the type of operative procedure suggested to her. I found it possible to salvage the sphincters of her rectum for which there is yet no available acceptable substitute, restoring intestinal continuity after excision of a large mid-rectal neoplasm. A decade or more went by, and the family and the patient had been very pleased with the result. Mr. A., though not a man of great means, had influential and affluent friends. He had been telling me he was anxious to do something for my department.

One day without announcement, Mr. A. came to my office bringing with him a Mr. B. After we had talked for perhaps twenty minutes of a number of generalities, Mr. B. said to Mr. A.: "Tell me, why are we here?" Mr. A. replied: "We have come to discuss your problem." Mr. B. said: "My problem? I have no problem!" "Yes, you have," replied Mr. A. "Well, what is my problem?" said Mr. B. "It is your money, Mr. B., your money!" Shortly thereafter a check arrived and a pledge in the amount of \$100,000. About ten days later, Mr. B. called to ask what progress had been made in our research. He wanted to be certain, I know, that we were good

stewards of his generous benefaction. Many years have gone by. Out of the discussion between Messrs. A. and B. has grown a continuing and extremely helpful source of research support. We are now in the midst of the third \$100,000 donation by that individual and his family. How wonderful it is to have a good and generous friend!

This discussion of one of your problems, I would like to believe, presages the growth of a great and important development in this hospital. It is in voluntary hospitals like yours, which address themselves seriously to responsibilities of teaching and research, as well as service, that the specialist of tomorrow will find good opportunities to prepare him for his life's work.

The struggles of your founders to place this hospital near or on the University Campus tells us much of the nature of their commitment to a closely linked University enterprise in your hospital. It is, I am certain, you will all agree, a worthy objective, which will determine in large measure the fate, promise, and directional growth of this institution. If everyone privileged to enjoy its opportunities will share the fervor and hopes which your founding fathers held for this hospital, their dreams and yours can and will come true.

There is a disposition among a certain group of our professional colleagues to credit the dreams of sleep as being more important than those of our waking hours. Some of you will recall the implied humor of such a conclusion as portrayed a few years ago in the *New Yorker*. Two middle-aged women crossing a pasture took refuge in a tree from a pursuing bull. Said one to the other: "If this were a dream, it might have real significance."

It is the dreams of men of hope and vision—dreams of men like your founders—which we want to share and trust.

In Dallas, Texas, many years ago, one Sunday morning over the radio I heard a minister tell this story:

He had gone out to call on a parishioner in the environs of Dallas. When the preacher came out, the small son of the sick father, whom the minister had visited, was admiring the minister's shining new Cadillac. The minister explained, somewhat apologetically: "My brother who has been very successful in oil gave me this wonderful car." Said the little boy: "When I grow up, Parson, I would like to be a brother like that."

The Mt. Sinai Hospital, I know, does not need to wait for those brothers to grow up. They are here.

TIGHTENING THE EDUCATIONAL BELT

The years ahead in many areas will mark great changes which will touch our own lives. One of these is education. We shall see I believe, a much needed and long overdue tightening up of the educational process beginning with the elementary school and continuing all along the line. We Americans have assumed a rather self-satisfied and superior air with regard to our situation in life. Recognition of the circumstance that we have lagged behind in certain areas of accomplishment has shattered our complacency. We have suddenly come to realize that competition at the international level needs a more serious appraisal of our educational responsibilities. Though the story of the hare and the turtle is reputedly a fable, it still has pertinent significance for us. The race is not always to the swift unless the goal is kept very definitely in mind.

HELP IN THE ACHIEVEMENT OF GOALS

In May 1948, I was privileged to be your speaker at a dinner in the Coffman Memorial Union on the campus of the University of Minnesota on the occasion when this Jewish community and American Friends of the Hebrew University in Palestine met to rededicate themselves to the task of helping rebuild the hospital there. On that occasion, some of you may remember, I dilated on the pleasure and satisfaction of service to one's fellow man. Many a person has found in that service, I am very certain, satisfying and acceptable resolution of his own problems. The gentle art of submission has taught many a man how to endure his lot when it cannot be enjoyed. He who spends his life in vain and selfish pleasures impoverishes his gift. But he who forgets himself and devotes his energies without stint to making the lot of others happier—that man enriches the precious gift of life, permitting it to blossom into the lovely ornament which it can become. This is the most important lesson which life has to teach us. Only he who strives to bring happiness to others will find it for himself. Was not this the vision of Sir Launfal?

Your prophet Micah's definition of the good life is the inscription which stands over the statue of religion in the Library of Congress. It reads: "What doth the Lord require of thee, but to do justly, to love mercy and to walk humbly with thy God"—a beautiful rule of life recchoed essentially in the precepts contained in the Sermon on the Mount. This motivation by many of your workers, if shared and remembered by all, will assure your effort of great success and its supporters of a source of enduring satisfaction and contentment.

The Treatment of Hyperthyroidism

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THE TERM HYPERTHYROIDISM will be used herein to denote a disease characterized by overactivity of the thyroid gland of such nature as to result in the net peripheral oversupply of those secretory products of the thyroid gland which control the metabolic rate of the organism. Clinically, this disease can be divided into 2 main syndromes: (1) that associated with a goiter essentially free of nodules and called Graves' disease or exophthalmic goiter, and (2) that associated with a nodular goiter and called toxic nodular goiter or toxic adenomatous goiter.

Graves' disease is most often associated with obvious evidence of hyperirritability and hyperkinesis, although the patient may not be very ill. This disease usually is of very abrupt onset and typically is encountered in young women. The thyroid in this disease may be insignificant clinically, but histologically it demonstrates a picture of diffuse hyperplasia with lymphocytic infiltration. Toxic nodular goiter, in contrast, is always associated with a definite goiter that can be either diffuse or localized but is always nodular. The patient may show any degree of hyperthyroidism from the most obvious textbook picture to one almost unrecognizable as hyperthyroidism, with the tendency being in the latter direction. As a rule, patients with this disease are older than are those with Graves' disease; still, they are more likely to be women than men.

Because of the obscure and confusing picture presented by many patients with toxic nodular goiter, the diagnosis is often overlooked early in the course of the illness. As a result, patients are apt to become quite ill with complicating problems, such as cardiac insufficiency, before the diagnosis is made. Consequently, such patients are often more seriously ill and closer to so-called thyroid crisis than are the more obviously hyperthyroid patients with Graves' disease. This difference is quite important in any discussion of therapy because the toxic nodular goiter patient often is in greater need of immediate treatment than is the Graves' patient.

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The interval from the start of therapy to the first signs of improvement may be critical.

TREATMENT

As of 1959, the treatment of hyperthyroidism has, for the most part, resolved itself into 1 mode of therapy: subtotal ablation of the thyroid gland. This is accomplished by administration of radioactive iodine (I^{131}) or operation. The latter is resorted to only after suitable preparation of the patient with an antithyroid compound. These compounds also have been suggested as alternative definitive therapy, but the unpredictable course of the patient after withdrawal of these compounds and the complications of continued usage have made most authorities consider such treatment programs second best.

Oral I^{131} has proved to be the most efficacious agent available for producing a remission in Graves' disease, which is the form of hyperthyroidism that responds best to I^{131} . The latter statement will be discussed later; the former is best illustrated by the experience of our thyroid clinic—a 100 per cent remission rate in the Graves' disease patients so treated. Even more impressive is the opinion held generally that, once rendered euthyroid, I^{131} -treated patients do not again exhibit hyperthyroidism. In our clinic there are only 2 instances that may prove to be exceptions to this statement. Yet, some caution in the interpretation of these statements is needed insofar as most patients treated in this way have been followed for less than fifteen years as of 1959.

The pattern of response to this form of treatment is predictable. As a general rule, the patient receiving I^{131} will experience no therapy-induced change in the clinical course of the disease for the first four weeks after administration of the first dose. On the negative side, this means that the patient fails to improve within the first month. On the positive side, it means that there has been no substantiated report that a patient has become suddenly worse because of an acute exacerbation of Graves' disease secondary to radiation effect. In particular, patients with serious disease, such as coronary or myocardial

insufficiency, are apparently not made worse by I¹³¹ therapy. Also, worsening of tracheal compression after use of this mode of therapy in patients with an already compromised airway has not been reported.

The first evidences of improvement, then, are noted in the fifth week. This initial improvement frequently is followed, within the next four weeks, by further change sufficient to erase most, if not all, of the remaining evidence of Graves' disease. An occasional patient will first show some clinical response only six or eight weeks after administration of the isotope. Others will continue to show improvement for eight, ten, or more weeks after the initial response has been demonstrated. Thus, several months may elapse before the maximum benefit to be expected from a therapeutic dose of the isotope has been experienced. It is easy to be misled, since many patients will experience improvement in clinical symptomatology within a few days or weeks after receiving the first dose. This benefit is almost invariably the result of ancillary therapy, such as bed rest or sedation, or the improved emotional outlook resulting from the patient's having consulted a physician who has promised to help. On the other hand, the patient may be worse within a few days or weeks, just as a result of the natural course of the illness.

Our practice is to treat the Graves' patient with a single dose of I¹³¹ calculated to produce a complete remission. In some clinics, the policy is to achieve this result with several doses. Contrary to the practice in many clinics, we calculate this dose in a rather arbitrary manner. The patient's thyroid is examined several times, and its estimated weight, arrived at by palpation and sometimes also by roentgenograms and scintigrams, is noted. The patient is then given 0.16 mc. of I¹³¹ per gram of estimated thyroid weight. This figure was arrived at post hoc by the thyroid group at Massachusetts General Hospital after several years' experience with this type of treatment. No heed is taken of the severity of the illness, the seriousness of complicating problems if they exist, or the initial I¹³¹ uptake value. The I¹³¹ uptake value is noted to be certain that it is indeed in the hyperthyroid range. Many more complicated methods of calculating the proper dosage exist and have been reported in the literature by their various proponents.

In our thyroid clinic, about 85 per cent of the patients treated with I¹³¹ for Graves' disease have experienced a complete remission of the disease after 1 dose. However, this remission may never actually be complete. The patient may be left with some goiter or some of the eye signs

of the disease and still be euthyroid, or cured. While these reservations may sound discouraging, we have never seen a patient whose residual goiter presented any problem. The course of eye problems in I¹³¹-irradiated patients is no less or more malignant than it is in patients relieved by one of the other modes of treatment. The 15 per cent of patients treated in this manner who did not become euthyroid after a single dose have still improved to some degree as a result of the initial use of the isotope. Most patients in this group have required only a second dose. An occasional patient has required a third and even a fourth dose. Additional doses have never been administered sooner than four months after the previous one, since improvement has been noted to accumulate for almost this length of time after a given dose of I¹³¹. Later doses are calculated by the same method, remembering that the gland weight used in the calculation is always that weight estimated at the time of the treatment dose in question.

It is important that these doses be given only after a diagnostic I¹³¹ uptake has been repeated and found to be in the hyperthyroid range. If the tracer value is in the normal or below normal range after I¹³¹ therapy, no further therapy is given, even if clinically indicated. Our experience has been that such patients will, in time, either continue to improve until they are euthyroid or demonstrate more thyroid hyperplasia and an elevated uptake. This type of "partially cured" patient has always been sufficiently improved by the radiation already administered to allow us to watch without risk.

It has been our fortunate experience that, up to now, we have not had to resort to a second mode of therapy in any patient once I¹³¹ treatment has been started. However, ancillary therapy with an antithyroid compound has been given after definitive isotope therapy when the condition of the patient has been so precarious that we have wished to render the patient euthyroid as rapidly as possible. Such additional therapy has always been started forty-eight or more hours after administration of the therapeutic dose of the isotope, so as not to interfere with its thyroïdal collection, and has been continued until the patient has no longer presented any grave clinical problems. Ancillary therapy has never appeared to compromise the predicted response of the patient to the isotope. However, some patients given combined therapy take an unusually long time to become permanently euthyroid. This is a reflection of the fact that some patients become euthyroid as a result of ancillary therapy only to regain enough evidence

of hyperthyroidism after the ancillary therapy is discontinued to require additional irradiation. Iodides have not been used for this in our clinic; instead, we have used 1-methyl-2-mercaptoimidazole (Tapazole) or propylthiouracil.

Patients with hyperthyroidism associated with a hyperfunctioning nodular goiter, in contrast to the patients just discussed, respond best to treatment with antithyroid compounds followed by subtotal thyroidectomy. The compounds used in our clinic in recent years are those mentioned in the previous paragraph. Tapazole is most frequently given every eight hours in 10- to 20-mg. dosage; propylthiouracil is similarly given in 100- to 200-mg. dosage. As a rule, toxic nodular goiter patients handled in this way become euthyroid within four to six weeks, but this period may stretch into six months, especially if the patient had previously been treated with iodides. These patients are never treated surgically until unequivocally euthyroid and reasonably recovered from other complicating problems. Once ready for operation, the patient is given, in addition to the antithyroid compound, iodides in small dosage for one to two weeks. The iodide is given as 5 drops daily of saturated potassium iodide or Lugol's solution. This treatment is almost invariably followed by a satisfactory clinical cure if the subtotal thyroidectomy is, technically, sufficiently complete. The incidence of recurrent disease should be negligible. Fortunately, with this type of treatment the patient rarely shows permanent hypothyroidism postoperatively unless the surgeon has, with great effort, performed a total thyroidectomy.

Operation predictably and quickly rids the patient of the physical mass of the goiter. This is important, since many patients with this disease have goiters large enough to pose a major cosmetic or mechanical problem for them. For reasons beyond surgeons' control, the nature of surgical treatment is such that one cannot discuss the experience at large institutions in the same way one can the experience with I¹³¹ treatment. There is no way to standardize an operation, much less to standardize surgeons. Furthermore, most medical centers do not have close enough cooperation between surgical and medical units to allow for adequate centralized follow-up of the thyroidectomized patient by the internists. Therefore, there are few, if any, meaningful statistical results of surgical treatment in our institution or around the country.

As discussed earlier, many toxic nodular goiter patients are sick enough with related or unrelated diseases to be considered poor surgical risks. Many of them are first diagnosed at an

advanced age. This means that, in some instances, the patient's reasonable life expectancy is only a few years and, in others, that the patient is unwilling to submit to operative intervention. Therefore, despite our reluctance to treat nodular goiter patients with I¹³¹, we actually do treat a fair percentage of them. In our experience, 1 and, occasionally, 2 doses of I¹³¹ have eventually rendered euthyroid all such patients in the previously noted interval of time. However, the total dosage involved, about 18 to 20 mc., has been roughly twice that required to help the patient with Graves' disease. We arrive at this dose by giving the patient 0.2 mc. of I¹³¹ per gram of estimated thyroid weight.

An important drawback is the fact that many such patients experience a posttherapy recurrence of hyperthyroidism after a variable period of euthyroidism. A cursory recapitulation of the histology of the multinodular goiter will explain why this happens. These goiters invariably present a mixed picture of hyperplastic, involutional, and normal glandular tissue; in addition, there are usually areas of fibrosis and even calcification. It is to be expected that the uptake of radioiodine will be spotty in such glands, being greatest in the areas of hyperplasia. It should also be remembered that, as a general rule, the degrees of radiosensitivity and hyperplasia are directly proportional. Consequently, much of the tissue in such a goiter is relatively undamaged by any particular dose of I¹³¹. Since hyperthyroidism, regardless of the type, is believed to be maintained, if not initiated, by anterior pituitary thyrotropin, it is to be expected that the nonirradiated, involutional areas of the gland will, in time, become hyperplastic. The disease can then easily again become clinically evident. Even when this sequence does not occur, the patient may be left with a goiter mass that presents a cosmetic or mechanical problem.

COMPLICATIONS

Complications of therapy and unwanted sequelae associated therewith are always important considerations in any discussion of treatment. No complications have been noted so far with I¹³¹ treatment of hyperthyroidism, except when relatively large doses of radiation are employed. In our clinic, we have not even seen radiation thyroiditis; but, then, we purposely have never given a hyperthyroid patient more than 20 mc. in a single dose. Complications of subtotal thyroidectomy are those of any operation involving an anesthetic and those attendant on surgery involving a highly vascular organ. The fact that this organ is located near a plexus

of nerves of the sympathetic chain adds to potential complications. If the patient is not completely euthyroid at the time of surgery, there is yet another possible complication—thyroid crisis.

Sequelae of the use of ^{131}I include one of practical importance—hypothyroidism—and one of theoretic importance—the induction of neoplasia in the irradiated tissues. In about 15 per cent of patients whose hyperthyroidism is treated with ^{131}I , permanent hypothyroidism develops. This is almost entirely limited to patients with Graves' disease. Sometimes, this eventuality does not become apparent until five or more years have elapsed from the time of administration of the last dose of the radioisotope. The theoretic sequela becomes more remote as time goes on. Many thousands of patients have received ^{131}I therapy since 1939. Actually, there is but one well-documented report of thyroid neoplasia which presumably first arose in an ^{131}I -irradiated thyroid gland after the irradiation. This report concerns two youngsters who had rapidly regrowing, hyperplastic goiters and who each received multiple doses of the isotope. On the other hand, unwanted sequelae of surgical treatment include hypothyroidism in about 10 per cent of the patients, vocal cord palsy, and permanent hypoparathyroidism. Hypothyroidism is no trouble to treat regardless of how it is induced, but vocal cord palsy and hypoparathyroidism are disabilities still only partially curable at best. The actual incidence of either of these truly frightening sequelae is unknown, but it certainly rises sharply as the caliber of the surgeon falls from the finest to the usual. It is indeed interesting that physicians always quote statistics reported by the world's finest surgeons when discussing the results of surgical therapy. Only a handful of patients are ever operated on by such men. Statistics involving the sequelae mentioned and applicable to the caliber of surgeon to whom most patients have access is not known with any certainty. As luck would have it, the opportunity for recurrent nerve damage and parathyroid removal is greatest in patients with large nodular goiters. So, again, circumstances tend to push the physician toward the use of ^{131}I treatment in the toxic nodular goiter patient.

The handling of "thyroid crisis" is a problem that always fascinates the student of thyroid disease, but the crux of the problem is not the handling of the crisis as much as the prevention of it. This unfortunate episode in the course of hyperthyroidism occurs as the result of either the inexorable progression of the disease to its logical end or of some trauma occurring to a

patient who is already hyperthyroid. There is no excuse today for any patient to reach the stage of crisis as a natural progression, and the situation rarely is encountered. There is equally little excuse for crisis to occur attendant to thyroidectomy. No patient need undergo premature thyroidectomy today, since there are no indications for emergency thyroid surgery to relieve hyperthyroidism. However, crises still have a minimal unavoidable incidence as the result of problems intercurrent in the course of hyperthyroidism. No one can prevent appendicitis from developing in such patients or from their having accidents. The treatment of such a catastrophe is still nonspecific. The patient should be heavily sedated and removed from noise and excitement. Fever is controlled as usual and the patient is fed intravenously if necessary. Most authorities advise the use of stable iodide at this juncture; often this is accomplished only by an infusion. Some suggest the administration of antithyroid compounds in relatively large dosage, and some think that cortisone-like steroids should also be given in pharmacologic doses. Regardless of how one views these plans of "specific" treatment, proof of the effectiveness of any or all of them is lacking. One must act in faith.

Earlier in this article it was implied that ^{131}I is the best treatment for every patient with Graves' disease. At the present time, this implication is accepted with reservation because of the risk involved in irradiating the thyroid of a young person. As noted earlier, the only conclusive report of ^{131}I -induced neoplasia so far encountered involves growing children, and there are theoretic reasons for this. Therefore, it is customary in our clinic to operate on all Graves' patients under the age of 25 years unless there are reasonable contraindications. Surgery is only performed after the patient has been rendered euthyroid by the administration of antithyroid compounds. This is usually achieved readily within four to six weeks. Another reason for the reservation is the problem of fetal thyroid irradiation. The fetal thyroid can capture ^{131}I more or less effectively by the twelfth week of gestation. For this reason, most clinics will not give ^{131}I to a hyperthyroid patient known to be pregnant. Such patients can fortunately be treated surgically quite effectively, and, if there is reason to avoid surgery, such a patient can be treated with antithyroid compounds throughout the pregnancy and with ^{131}I after parturition.

There has been a good deal written in the literature about exophthalmos, and much of it misleading. For example, some authors imply that it is inadvisable to use surgery as treatment for

hyperthyroidism in a patient who has unusually severe exophthalmos, yet the best therapy for such patients remains treatment of the hyperthyroidism by any means. The incidence of progression of exophthalmos following therapy for hyperthyroidism is the same for all types of effective treatment; it is minimal. Unfortunately, the entire problem of treating exophthalmos, presumably always of anterior pituitary origin, is poorly understood. A discussion of this topic is beyond the scope of this article.

CONCLUSIONS

There is every reason to be pleased by the present status of the treatment of hyperthyroidism.

The abnormality is reasonably well understood in terms of the pathologic physiology involved, even though the etiology is only poorly understood. Diagnosis can be approached scientifically and effectively. There are therapeutic tools available which will induce a satisfactory remission of the disease in about 100 per cent of all patients. In addition, there is reasonable hope that therapy will soon be entirely medical and, as such, will not be followed by any important unwanted sequelae or attended by any undue risk or even disfigurement. Finally, effective therapy will be available to all where they live. Few ills of man have been dealt with as satisfactorily.

RADIOACTIVE PHOSPHORUS (P^{32}) has a rapid rate of uptake by red cells with thyrotoxicosis and a slow rate with myxedema. When treatment with thyroid or antithyroid drugs invalidates tests with radioactive iodine, P^{32} is especially useful in diagnosis. Serial determinations of P^{32} uptake are an accurate index of reaction to treatment, and the patient need not be exposed to ionizing radiation.

In some conditions, P^{32} uptake is not specific for thyroid function. Patients receiving large doses of corticosteroid usually show abnormally high values, and some cardiac disorders will cause low values.

For each test, 15 cc. of venous blood is transferred to a heparinized flask. A 2-cc. sample is used for a duplicate measurement of the hematocrit, and the remaining 13 cc. is poured into a tube containing 0.4 μ c. of P^{32} . After being shaken gently, the tube is placed in a water bath of 37° C. During the incubation period, a mixture of 95 per cent oxygen and 5 per cent carbon dioxide is bubbled slowly through the blood. After forty to fifty minutes and again after seventy-five minutes, a 4-cc. sample is withdrawn and divided into 1- and 3-cc. portions. The small portion is completely hemolyzed by the addition of 9 cc. of a solution of sodium chloride and sodium biphosphate, together with a few grains of saponin. After the 3-cc. portion has been centrifuged at 2,500 r.p.m. for five minutes, 1 cc. of plasma is withdrawn and diluted with carrier solution of sodium biphosphate to a final volume of 10 cc. The radioactivity of each sample is measured in a liquid counter.

Total radioactivity as measured in the hemolyzed specimen remains constant. Thus, the rate of P^{32} uptake by red cells can be calculated by measuring changes in plasma P^{32} content, taking into consideration the previously determined hematocrit. The procedure must be conducted within three hours, since pH alters and hemolysis occurs after that time.

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Vulvar Adenocarcinoma of Apocrine Gland (Paget's Disease of the Vulva)

Report of a Patient

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IN 1874, Sir James Paget¹ described a lesion of the breast now commonly known as Paget's disease of the nipple. It is a chronic, eczematoid, finely granular, and ulcerative skin involvement of a low-grade carcinomatous nature. He alluded to the possibility of its location in other sites. Paget's disease of the breast is not common, but its occurrence in the vulva is exceedingly rare.

Adequate reviews^{2,3} and separate case studies leave some question as to the total number of patients reported in the literature. The most recent report, in February 1955, sets the number at 23.³ One patient, however, had been presented on two separate occasions.⁴ Several case reports since 1951 were also omitted.⁵⁻⁹ Considering this, we believe there have been 28 reported incidences of vulvar Paget's disease; our patient constitutes the twenty-ninth.

CASE REPORT

Mrs. E. S., aged 62, was first seen in February 1954. At that time, she complained of burning and itching of the vulva of three years' duration. This had become worse in the past four weeks, so that she had again sought medical care. Various ointments, lotions, and other local treatments over the past years had given but temporary and incomplete relief. The labia majora were edematous, and the entire apposed surfaces presented a brilliantly reddened, sharply demarcated, moist and weeping lesion. White, adherent plaques of varying size were scattered over the involved area. The labia minora were obliterated and the clitoris "hooded." Surface cultures became strongly positive for *Candida albicans*. Since the clinical impression was that of monilia-

sis vulvitis superimposed upon kraurosis vulvae, conservative treatment was instituted. Temporary symptomatic relief followed, but there was little change in the gross appearance of the vulva. Therefore, in April 1954, a complete vulvectomy with wide excision of the labia majora was performed.

The gross specimen consisted of the completely amputated vulva measuring 12 x 10 x 2 cm. The vulva mucosa near the introitus was tan, smooth, and shiny with numerous irregular, slightly elevated plaques near the periphery. Microscopic examination from several blocks of tissue revealed the characteristics of leukoplakic vulvitis. However, the deeper portions of the squamous epithelium on the surface and of the exaggerated rete pegs were extensively invaded by large, neoplastic cells with clear, foamy cytoplasm and hyperchromic nuclei. These were typical in appearance and arrangement with the much described and well known "Paget cells" (figures 1, 2, and 3). In one area (figure 4), a sweat gland and its ducts showed similar neoplastic involvement. No lymph channel invasion was found. In one block, from the left upper quadrant of the removed vulva, the lesion appeared to extend to the edge of the incision. In June 1954, a left superficial and deep inguinal node dissection with excision of this segment of skin was done. This was followed in September by a right superficial inguinal node dissection. These tissues failed to exhibit neoplastic elements. The patient did well following each surgical procedure and had no gross or symptomatic evidence of recurrence during the immediate postoperative follow-up.

COMMENT

It is not our desire to entertain an extended discussion of the clinical features, morphologic characteristics, or pathogenesis of vulvar Paget's disease. These have been adequately considered by others.^{2,3,6,8-10} In 1937, Weiner² proposed "that Paget's disease of the skin is due to intra-epidermal metastases of carcinoma of the apocrine sweat glands." Subsequent studies have

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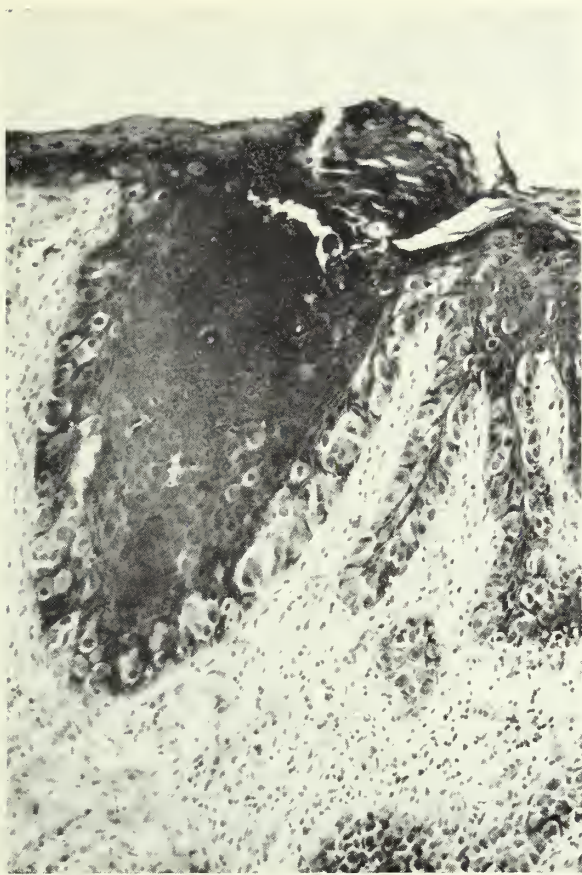


Fig. 1. A representative area of the superficial vulvar epithelium showing the exaggerated rete pegs with the secondary invasion of the lower epithelial layers by the typical Paget cells. Considerable round cell infiltration of the lamina propria is noted. ($\times 50$)

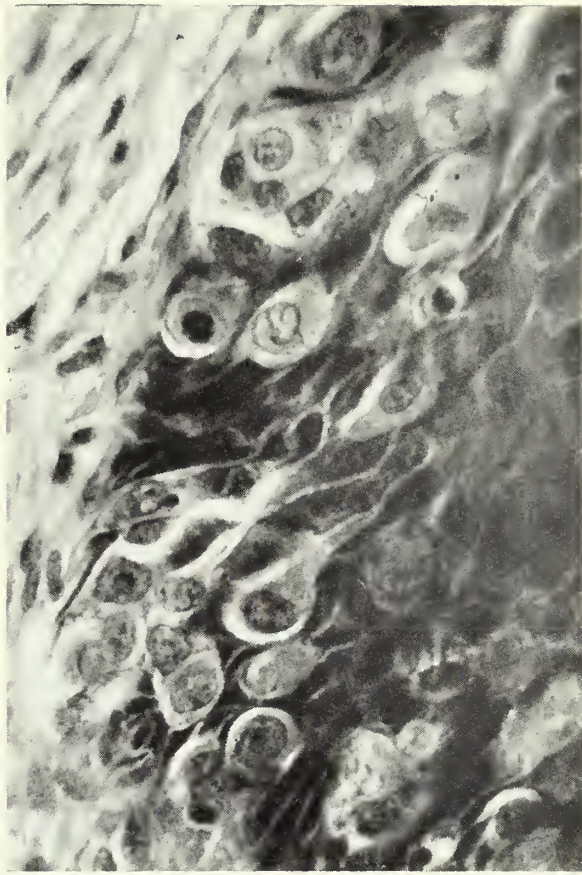


Fig. 2. This represents a higher power of the left of the large rete peg shown in figure 1. Several mitotic figures are noted. The clear, foamy cytoplasm of the typical Paget cells is seen. ($\times 100$)

supported such a view. Fortuitous sectioning of the specimen from the patient herein considered revealed one such gland. Further blocking might well result in the discovery of others. Dockerty and Pratt⁶ have made it quite apparent that the involvement of the superficial epithelium is secondary in time of appearance to the neoplastic changes within the deeper, underlying sweat glands.

Sweat glands elaborate their secretions by one of three different methods. That of apocrine glands is attended with loss of a part of the cell's cytoplasm into the lumen. Apocrine glands also pass through cycles of secretory activity.⁹ They are found only in the axillary, the external genital, and perianal skin areas. It has long been conceded that the mammary acini and ducts have been derived from modified apocrine sweat glands. Plachta and Speer⁹ have clearly shown that the "pagetization" of the vulvar epidermis

extending from carcinoma of the apocrine glands parallels the findings in ductal carcinoma or Paget's disease of the breast according to Cheate and Cutler.¹¹

The author is cognizant that criticism might be directed because preoperative biopsy was omitted in this patient. However, others have warned of the difficulties attending accurate diagnosis from biopsy material when dealing with so complex a structure as the vulva. Noteworthy among these is the report of a necropsy by Plachta and Speer.⁹ Vulval biopsies of their patient in 1935, 1939, and 1948 revealed only innocuous lesions, but four years later she died of extensive metastases arising from an adenocarcinoma of the vulva of apocrine gland origin. Those authors assert that the lesion was present at the time of the previous biopsy, but that its true nature missed because of inadequate material. Although preliminary biopsy was consid-

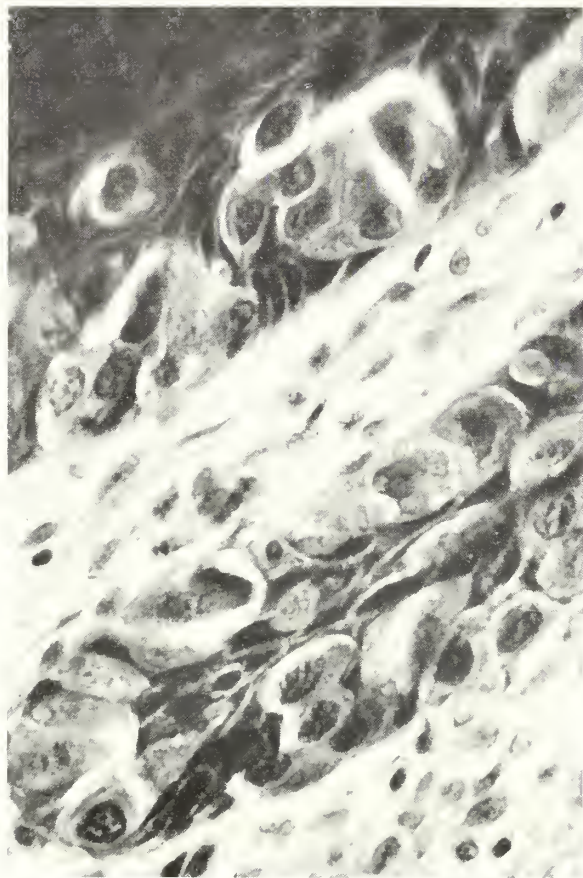


Fig. 3. Another higher power illustration from the right of the large rete peg previously mentioned in figures 1 and 2. The more narrow rete pegs have been almost entirely replaced by the neoplastic cells. ($\times 100$)

ered, the uniformity of the lesion's appearance left us in a quandary as to what regions should be biopsied. It was felt that satisfactory symptomatic relief could come only from a complete vulvectomy. With this predominantly in mind, it was elected to carry out definitive treatment in the form of wide excision of the entire vulva, having advised the patient that bilateral lymphadenectomy might be necessary later should a neoplasm be discovered. Even though this method was selected in the presence of an unknown carcinoma, it is believed that such a course proved most expedient. This line of reasoning seems to have popularity among those who have recently reported their methods of handling a patient under similar circumstances. At the same time, we are not at all adverse to obtaining a biopsy of isolated or circumscribed tumors whenever they involve the vulva.

Further excision of the perineal skin was imperative when the discovery of extension to the

edge of the incision was made on microscopic examination. Although, by gross appearance, adequate excision had been done, it is apparent that microscopic extension alarming distances beyond the detectable line of demarcation is possible. Frequent recurrence after complete vulvectomy has been noted by others.^{3,6,8} Obviously, adequate study of the specimen must be done to substantiate complete excision; for only when this exists can we hope to obtain encouraging clinical results.

SUMMARY

A patient with vulvar adenocarcinoma of apocrine gland origin (Paget's disease of the vulva) has been reported. A short commentary on the nature of this condition and the difficulty of obtaining accurate diagnoses from biopsies followed. Suggestions regarding the definitive treatment of similar lesions in other patients have been given.

Since this report was prepared, several other isolated case studies have appeared in the literature.

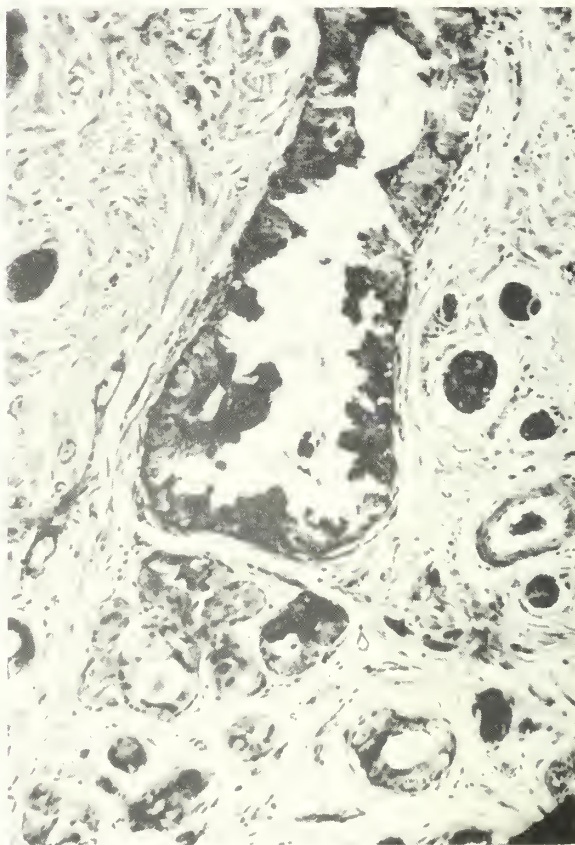


Fig. 4. The apocrine sweat gland and duct involved by adenocarcinomatous changes. ($\times 50$)

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INCISION OF the cervix can be an aid to management of some types of dystocia and fetal distress if the correct indications, conditions, and technic are observed. In such cases, fetal mortality should be no higher than with other operative deliveries. In addition, manual dilation of the cervix is less likely when the cervix is incised.

Hysterostotomy is not meant to replace abdominal delivery; requisites for each operation are distinct and separate.

Incision of the cervix is warranted in the following instances:

- When cephalopelvic proportion is normal. In such cases, x-ray pelvimetry should be used to help judge adequacy of the pelvis.
- When the patient is not suffering from placenta previa.
- When the presenting part is engaged at either +1 or +2 station.
- When the membranes are ruptured.
- When the cervix is dilated 5 cm. or more and is completely effaced.
- When labor has been given an adequate trial, with sufficient attention awarded to hydration, rest, stimulation, and antibiotic therapy.

In order to facilitate delivery, a deep episiotomy is done before the cervix is incised. So that the greater branches of the uterine artery may be avoided, the Dührssen incisions are made between ring clamps, under direct vision if possible, at 10, 2, and 6 o'clock, extending from the external os to the fornix. Inadequate incisions may cause laceration in the lower uterine segment, resulting in hemorrhage and shock. All 3 incisions must be made in most patients, particularly if the cervix is dilated about 5 cm. However, if dilation is 8 cm., 1 incision often causes complete retraction of the cervix.

After the fetus and placenta have been delivered, the cervical incisions are repaired with interrupted 2-0 chromic catgut sutures, followed by a continuous imbricating suture of the same material. The second suture aids healing, helps suppress bleeding, and prevents the cervix from adhering to the wall of the vagina.

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Maxillofacial Trauma

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EMERGENCY TREATMENT of maxillofacial injuries begins at the scene of the accident with control of hemorrhage, maintenance of an adequate airway, and treatment of shock. Of utmost importance is the prevention of oral secretions and blood drainage into the oropharynx. This can be accomplished by placing the patient in a prone or side position. Emergency tracheotomy is sometimes required in severe facial fractures with obstruction to the airway. In addition to providing an adequate airway, it will allow for greater ease in administering anesthesia during surgery.

An accident which can produce severe facial trauma is capable of producing injury to the central nervous system. Before any definite treatment of facial injury is undertaken, evaluation of brain damage by a neurologist or neurosurgeon is essential.

It is necessary to have the patient alert and cooperative in order to obtain good facial bone x-rays. If fractures of the middle third of the face are suspected, x-rays should be taken in the stereo Waters or oblique Waters position. X-rays of the nasal bones should be obtained if nasal deformity exists to determine whether or not this is a recent injury. Study of the mandible should always include the condylar heads and necks, since a blow to the symphysis region is capable of producing a fracture dislocation in this area.

The best postoperative results are obtained when the fractures can be reduced within twenty-four to forty-eight hours. Because central nervous system injuries and internal injuries take precedence, this is not always possible. It is extremely difficult to properly align the facial bones after ten days or two weeks. If a considerable amount of time has elapsed, reconstructive surgery with the use of bone or cartilage grafts may be required to correct asymmetrical facial contours.

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During a one-year period—November 1, 1958, to November 1, 1959—154 cases of maxillofacial trauma were seen. Of this number, 116 cases, or 75 per cent, were due to automobile accidents (table 1). It is interesting to note that the greatest number of accidents, 34, or 29.3 per cent, occurred in the 11 to 20 age group and the second largest number, 31, or 26.7 per cent, occurred in the 21 to 30 age group. Eighty-eight persons, or 75.9 per cent, were passengers as compared to 25, or 21.5 per cent, who were drivers and 3, or 2.6 per cent, who were pedestrians. The most common injury was facial laceration, with an almost equal distribution between facial lacerations with associated facial fractures and facial fractures only. From the histories obtained, none of the patients was wearing a safety belt. The remaining 38 cases of trauma, or 25 per cent, were caused by industrial accidents, home injuries, fights, tractors, carnival rides, homemade bombs, boating, football, and golf.

The contusions and abrasions were first cleansed with saline and solvents. Where embedded foreign material in the dermis was present, an abrasive technic using fine sandpaper was carried out. An ointment pressure dressing then was applied to the area. The lacerations were cleansed meticulously and the ragged edges were sharply recut before the lower derma was sutured with fine catgut. The skin was sutured with 6-0 silk interrupted sutures. The sutures were removed early, and fine mesh gauze and collodion dressings were applied.

When avulsion of the skin was present, it was corrected by the use of either a rotation or sliding skin flap. None of these patients presented a loss of skin large enough to warrant the use of an immediate split-thickness skin graft. Where an associated bone fracture with facial lacerations occurred that required an open reduction and wiring of the fragments, an attempt was made whenever possible to do the procedure through the laceration rather than through an incision which would result in a second scar.

Fracture of the nasal bones and fracture dislocation of the septum were reduced and fol-

TABLE 1
MAXILLOFACIAL TRAUMA RELATED TO AUTOMOBILE ACCIDENTS
116 Cases from November 1, 1958, to November 1, 1959

Age group	Males	Females	Driver	Passenger	Pedestrian	Laceration	Facial laceration with facial bone fracture	Facial bone fracture	Cases	Per cent
1 to 10	8	6	—	12	2	12	1	1	14	12.1
11 to 20	14	20	5	29	—	23	7	4	34	29.3
21 to 30	18	13	7	24	—	23	6	2	31	26.7
31 to 40	5	11	4	12	—	9	2	5	16	13.8
41 to 50	2	5	2	5	—	3	3	1	7	6.0
51 to 60	4	5	5	4	—	2	3	4	9	7.8
61 and over	2	3	2	2	1	—	2	3	5	4.3
Total	53	63	25	88	3	72	24	20	116	
	45.7%	54.3%	21.5%	75.9%	2.6%	62.1%	20.7%	17.2%		

lowed by the insertion of ointment packing into the nasal cavities with the application of a metal nasal splint.

An open reduction using Gillies'¹ approach through the temporalis fascia was most commonly used for reduction of fractures of the malar compound and the zygomatic arch. If the fracture did not remain in its normal position after reduction, it was necessary to wire the malar compound at the zygomaticofrontal suture line. When a fracture of the orbital floor had been sustained, a Caldwell-Luc procedure with insertion of ointment packing into the antrum was done. The ointment packing was left in place for a period of three weeks.

The method of treatment for fractures of the mandible and maxilla was dependent on several factors—whether the patient was edentulous, where the fractures were located, and whether both the maxilla and mandible were fractured.

When the patient had either a fracture of the maxilla or mandible and teeth were present, arch bars were placed on the upper and lower jaws and wired to the individual teeth with stainless steel wire. Rubber traction was applied to the arch bars to keep the jaws immobilized. This was continued for six weeks.

For fractures of the body of the mandible in edentulous patients, an open reduction and wiring procedure was accomplished. In the case of a fracture dislocation of the condylar head and neck of the mandible, a Kirschner wire was passed through the symphysis region. A plaster headcap with an incorporated wire was constructed, and rubber traction was applied to the

Kirschner wire for four weeks. No open reduction and wiring was done at the site of the fracture dislocation of the condylar head and neck of the mandible in patients with or without teeth.

Internal wire fixation was used as recommended by Adams²,³ when both fractures of the mandible and maxilla were present and the patient was not edentulous. In this procedure, a stainless steel wire was inserted in a drill hole at the zygomaticofrontal suture line, passed under the malar bone down through the buccal sulcus, and attached to the arch bars. The wire was removed after five weeks. This eliminated wearing a cumbersome plaster headcap.

It is possible to predict the prognosis of facial lacerations almost immediately. Those which are parallel to Langer's lines will result in a fine line scar, while those running in a perpendicular direction will have a tendency to widen or become hypertrophied. Revision of these scars may be required at a later date, usually six months to one year after the time of injury.

SUMMARY

1. A total of 154 patients with maxillofacial injuries was seen during a one year period—November 1, 1958, to November 1, 1959. Of the patients, 75 per cent received injuries as a result of automobile accidents, and 56 per cent of injuries occurred between the ages of 11 and 30 years. Passengers constituted 75.9 per cent of the patients, and none gave a history of using a safety belt.

2. Because of the danger of injury to the central nervous system as a result of severe facial

injury, no definite treatment should be undertaken in such cases until brain damage has been evaluated by a neurologist or neurosurgeon.

3. It is necessary to have the patient alert and cooperative in order to obtain good facial bone roentgenograms.

4. When a facial bone fracture with facial laceration occurs and an open reduction and wiring procedure is necessary, an attempt should be made to accomplish this through the laceration to avoid making a surgical incision which would result in a second scar.

5. The best cosmetic results are obtained when

treatment can be initiated twenty-four to forty-eight hours after injury. A delay of ten days to two weeks for facial bone fractures makes it difficult, and on occasion impossible, to reduce the fractures. As a result, bone or cartilage grafts may be necessary to correct the asymmetry of facial contours.

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CAUDA EQUINA compression, most commonly caused by central prolapse of a lower lumbar intervertebral disk, causes characteristic sensory, motor, and visceral disturbances. The majority of patients have suffered from low back pain, leg pain, numbness, or tingling paresthesias for months or years before symptoms of cauda equina compression are noticed. Back injury or strain due to twisting or lifting is often involved. Frequently, patients recall 2 distinct traumatic events—one preceding the onset of back and leg symptoms and the other heralding cauda equina compression. Symptoms of compression usually develop rapidly and are frequently asymmetric.

Motor manifestations of compression include muscle weakness below the knees; diminished or absent ankle reflexes; and atrophy, loss of tone, and weakness of the gluteal muscles. Sensory manifestations include impaired skin sensation in the saddle area and pain, numbness, or tingling paresthesias in the legs. Visceral involvement may cause difficult micturition or urinary retention, loss of bladder and rectal sensation, constipation, loss of rectal and anal sensation, and sexual impotence.

Manipulation should be avoided, as it is known to be dangerous. Laminectomy should be performed as soon as the diagnosis is made. Delay in operation causes irreversible nerve damage and incomplete recovery of function.

Cauda equina compression does not directly endanger life but, if left untreated, can cause ascending urinary infection due to bladder paralysis and decubitus ulcers resulting from impaired skin sensation. Uremia or septicemia resulting from compression may lead to premature death.

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Emotional Factors with Eye Patients

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A PATIENT seeking professional help for an eye complaint may be labeled as having (1) a purely physical or organic condition, (2) an organic condition with an inappropriate emotional reaction to this illness, or (3) a purely emotional condition with no organic features. The percentage of patients with eye disorders involving emotional conflict to some degree is very high—probably about 75 per cent. Much is said and written about the organic conditions. Our orientation is primarily organic, and so the emotional aspects of a condition are not recognized or are ignored for lack of interest. However, if we wish to give weight to the relative frequency of the “emotional ease” among eye patients, we must conclude that more time and energy should be spent in studying these cases, attempting to find the cause, and effecting a solution if possible.

The patients' attitudes regarding refractive errors, wearing and not wearing glasses prescribed, levels of illumination, and use of the eyes, as the people relate them to organic eye health, are important factors in producing eye symptoms. These feelings result in abnormal, unrealistic, and unjustified eye-consciousness and this, as we see daily in practice, often leads to symptoms which are disabling to some degree and are unprofitable financially. An attempt will be made to describe some of these attitudes in more detail.

PATIENT ATTITUDES

First, and probably most prevalent, is the exaggerated feeling regarding refractive errors and

wearing glasses to correct them. To a high percentage of our population, any degree of farsightedness or nearsightedness means progressive eye disease, and a bit of astigmatism seems akin to smallpox or some similar disease. When these conditions are explained as anatomic variations of the normal and are compared with normal variations in other parts of the body, such as hand and foot sizes, a different feeling is stimulated and a healthier beginning is made for those who should wear glasses.

When glasses are prescribed, we are confronted with another common feeling which also merits attention. More often than not, the wearing of glasses is thought to be a preventive measure against the development of some disabling eye condition. This, we must agree, does exist with some cases of strabismus and anisometropia (refractive imbalance in both eyes), but these situations are relatively rare and it is not reasonable to saddle the great majority with a potential hazard for the needs of a small minority. This majority very much needs to have a detailed explanation of why glasses are prescribed. One or all of the following conditions must be fulfilled by wearing glasses:

1. Improved vision (distance, near, or both).
2. Greater comfort in visual activity.
3. Greater visual efficiency as a result of fulfilling one or both of the foregoing conditions.

From these points, we can make considered predictions in regard to the general results of wearing glasses all of the time or part of the time, but we do the patient an injustice if we insist upon his wearing glasses beyond the point where he himself can recognize benefits from doing so. When the patient understands these simple and explainable points about glasses, he

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or she is much less likely to develop exaggerated and unwarranted feelings about eye disease.

Another common exaggeration concerns the relationship of levels of illumination to eye health. The average person has been led to believe that a specific light intensity is right for a given task and that higher or lower intensities cause physical eye damage. This feeling is being broadcast by our school teachers, lighting engineers, and a good many eye professionals—not to exclude some ophthalmologists. Apparently it is easier to state and believe that "poor light is hard on one's eyes" than it is to explain and rationally understand that an optimum level of illumination relates only to the efficiency and comfort of a given task and not to the physical well-being of the eye. When a person believes that there is a physical organic connection with lighting, an element of anxiety is very often introduced. If uncontrollable levels of illumination are then encountered, he develops symptoms and often, as we see in practice, goes to much trouble and expense to "save his eyes from the light." Related to this subject is the time-worn advice to "read with the light coming from over one's left shoulder." This is good, as it eliminates the light source from the reader's field of vision and prevents shadows on the reading material for the right-handed page turner. However, to my knowledge, the left-handed page turner has never been advised to direct his light from over the right shoulder. Our school children are being directed on this matter daily. If the advice is worth giving at all, the reasoning behind it should be included and the exceptions to the rule would probably be understood and practiced.

To continue with the subject of light sensitivity, we must consider the great lot of people having the "glare" problem. These people use the term "glare" to mean any situation in which the lighting is uncomfortably excessive to them. They also have the feeling that this "glare" does physical damage to their eyes. Bright, sunny days, on-coming car headlights, and innumerable industrial work situations are responsible for the bright lights so troublesome to some persons. The people so afflicted are wearing a great variety of tinted lenses and sunglasses. Many or most of these lenses are being worn night and day in all levels of illumination. Many professional eye practitioners are advising and prescribing them without adequate explanations. The problem that these people have is an abnormal sensitivity, or low threshold, to normal variations in light intensity. This is the large group with psychogenic photophobia and does

not include the true photophobia associated with iritis, keratitis, and so forth. When these psychogenic photophobic people are prescribed the tinted lenses, their abnormal sensitivity is nurtured and they truly believe that the lenses "save their eyes." Also, the irrationality of the use of tinted lenses is fortified by these points:

1. Tinted lenses filter out about 3 to 8 per cent of the light. This is "a drop in a bucket" on an average sunny day when the light can be reduced 75 to 80 per cent without loss of visual acuity.

2. Tinted lenses are usually worn day and night—at all levels of illumination. Instead of the tint during periods of artificial lighting, why not use a weaker light bulb for homework or reading?

3. Tinted lenses do reduce the light from the on-coming car headlights by the usual 3 to 8 per cent, *but* they also reduce, by the same percentage, the light necessary for the wearer to proceed safely on his side of the road.

4. Tinted lenses have virtually no advantage in filtration of ultraviolet radiation over the common, clear quartz lenses.

These are some of the interesting and instructive points on the subject of tinted lenses that all people with sensitivity problems are entitled to know before these lenses are worn. When the higher grades of lens "coloring" are used, as with darker sunglasses, there is less validity to the foregoing arguments because a much higher percentage of the light is being filtered out. However, here, too, the person should be aware of the fact that the higher levels of illumination, without the sunglasses, do no physical damage to the eye. The relatively rare exceptions to this are the infrared retinal burns which occur from gazing at the sun for a long period or the intense light and heat from a blast furnace.

Another area in which people commonly display abnormal eye-consciousness is with the "use of the eyes." Even with best correction in glasses, or with no need for glasses and no ocular disease, a feeling is often expressed that eyes are "saved" by shorter periods of use. This feeling is currently being encouraged by certain professionals, particularly in relation to nearsightedness as it normally progresses in the growing child. As there is no real basis for a relationship between "use of the eyes" and any eye condition, this practice must be deplored. The psychologic effects are damaging and the curtailment of visual activity is a roadblock in any person's life; an example is the child who is poorly advised to cut down on reading because of normally progressing nearsightedness.

Related to the subject of the use of one's eyes is the attitude which many people have about viewing television. They feel that long periods with a television set are not good for the eyes. Many parents use this argument, sometimes believing it and sometimes using it without conviction, to instill a fear in their children in an attempt to better control their habits of viewing television. There should be better reasons for not "escaping" to the television set which are more reasonable to the child and less apt to stimulate an abnormal eye-consciousness.

The emotional response that people display with organic eye disease may also be inappropriate and show a pattern for the general group. For example, people usually feel that a diagnosis of cataract is far more ominous than that of glaucoma, retinal detachment, or macular degeneration. The case is generally the opposite, but eye practitioners usually keep this point in mind when early signs of cataract formation are seen. The words "lens opacities" are used until the lens has become cataractous to the point of

necessitating surgical removal. Whenever the word "cataract" is first used, a good bit of preparation and explanation are often necessary to avoid emotional upheaval.

When serious eye disease results in total or near total loss of vision, the emotional response is often incapacitating and seemingly appropriate. With these fortunately infrequent cases, we continue to need the help of psychiatrists, psychologists, and social workers for rehabilitation.

CONCLUSIONS

We can say there are many emotional factors in the production of eye symptoms serious enough for a great number of people to spend much time and money seeking solutions. Many of them are founded in prevalent attitudes of "the times." In dealing with these attitudes as medical practitioners, we must weigh the disadvantages of overconcern, or abnormal eye-consciousness, with the disadvantages of no concern and arrive at a probable middle-of-the-road course of action, which is the ideal.

FATIGABILITY of the extraocular muscles is often the only manifestation of myasthenia gravis. Onset is usually insidious, and the most obvious weakness is a drooping eyelid or transient diplopia. Occasionally complete external ophthalmoplegia is produced.

The facial muscles in the orbicularis oculi region are almost always involved, creating the typical myasthenic facies—a tendency toward obliteration of the normal folds and a lack of compensatory furrowing of the forehead with ptosis. Because extraocular muscle paresis is a frequent sign of intracranial aneurysm, this condition should be considered in the differential diagnosis.

ACTH, in a total dosage of 600 mg. divided into single doses of 20 mg. every six hours, is effective in reducing or eliminating extraocular muscle pareses for three to twelve months. Although relapse is expected, it is usually relatively slight. The dosage may be repeated but, in most cases, not until at least six months have elapsed.

Although therapy with neostigmine or pyridostigmin (Mestinon) is usually satisfactory for generalized disease, it is often only partially effective in relief of ocular pareses.

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Radiographic Procedures in Examination of the Liver, Gallbladder, and Bile Duct System

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THE PURPOSE of this paper is to give an orientation of the various roentgen methods available, with particular emphasis on facilities in our smaller hospitals. No attempt will be made to cover every detail in this large field. Current technics and recent advances in the field of radiology of the liver, bile ducts, and gallbladder will be mentioned, with emphasis on some of the more practical aspects in everyday practice.

LIVER

Radiographic examination of the liver now includes several specialized methods but until fairly recently consisted only of evaluation of size and shape on plain film studies. We are all aware that assessment of liver size is often erratic. Downward displacement of the lower edge determined by palpation or a film is sometimes misleading. It is generally understood that, for an accurate evaluation of liver size, information regarding its upper border, as manifested by the position of the diaphragm, is required. It is not as commonly appreciated, however, that constitutional variations in shape and position may alter the significance of clinical findings. In a stocky person, the liver tends to have a more transverse position and is considerably thicker from front to back than in the more lanky person, in whom it is more elongated and may lie mostly in the right abdomen with its tip down to the iliac crest. Thus, as well as correlating with the position of the right diaphragm, one must judge the type of liver to be expected in the various types of body build.

Demonstration of liver parenchyma and evaluation for detection or exclusion of local deformities, such as those which occur with neoplasm, abscess, or cyst, requires specialized study. Several technics are available, most of which are cumbersome, expensive, and not in-

nocuous enough for daily use. These include pneumoperitoneum, portal venography, arteriography, and the use of radioactive materials. Since these methods are not ordinarily used in smaller hospitals, we are going to say no more about them.

BILE DUCTS

The most significant advance in the study of the biliary tree in recent years concerns the development of an intravenous method of radiographic visualization of the intra- and extrahepatic biliary systems. The method and material used were developed in Germany. The trade name of the material in this country is Cholografin. This material given intravenously is mainly excreted by the liver, but, even in the presence of a normal liver function, 10 per cent is excreted by the kidney. The quantity excreted by the kidney increases with liver damage.

After injection, the dye begins to enter the intrahepatic, large hepatic, and common bile ducts within even as short a time as ten minutes. The common duct slowly becomes visible with optimum concentration in twenty to sixty minutes, gradually fading after that time. If the gallbladder is present and the cystic duct is patent, the gallbladder also fills. Its time of filling is more variable; it may be visible in twenty minutes or not for two hours. The filling of the gallbladder by the opaque dye does not demonstrate gallbladder function, as, with this material, the concentrating function is the property of the liver. Thus, as far as the gallbladder is concerned, the intravenous dye shows only structure and contents, if any.

It is reported that visualization of the duct system can be obtained in 80 to 85 per cent of cases. The visualization rate depends on the selection of cases, since, as the degree of liver damage increases, the rate of visualization decreases. If the test is applied indiscriminately, the visualization rate will be low; if it is reserved only for patients without jaundice or depressed liver function, the visualization rate will be high. Since it may be precisely the patient with mild

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jaundice and borderline liver function in whom the test will be most helpful, it is obvious that too much emphasis can be placed on the rate of visualization. There is no absolute line of demarcation where the probability of visualization can be predicted. However, it is true that there is little justification in attempting this study in patients with serum bilirubin above 5 mg. or in the presence of a Bromsulphalein retention of over 40 per cent.

In clinical application, the intravenous cholangiogram has been used mostly in the evaluation of the duct system in the patient who has had a cholecystectomy. By this method, one attempts to demonstrate partial obstruction of the bile ducts secondary to stones, incomplete duct strictures, stenosis of the sphincter of Oddi, or the possible presence of a cystic duct remnant. The interpretation of the intravenous cholangiogram under these circumstances is based on the following factors:

1. *Size of the common duct.* There are many reports in the literature which indicate that there is a fairly wide range in sizes. In the large series of cases analyzed after cholecystectomy, the sizes of the unobstructed ducts varied from 3 to 15 mm., whereas the diameters of the obstructed ducts ranged from 8 to 30 mm. The range, then, from 8 to 15 mm. is one of considerable overlap in differentiation of obstructed from unobstructed ducts. Since 50 to 60 per cent of visualized ducts will fall into this range, it is apparent that the mere size of the duct will not permit a distinction of the presence or absence of obstruction.

2. *Duration of opacification of the common duct.* In the unobstructed duct, the densest opacification is usually obtained in one hour and gradually fades over the second hour. It is possible, therefore, to make a diagnosis of partial obstruction on the assumption that the density of the dye does not decrease in two hours from its maximum at one hour.

3. *Presence of filling defects.* These shadows are often difficult to distinguish from overlying bowel shadows. However, one can judge by the constancy of their position in the various projections and on second examinations. Planigram studies may help, also.

4. *Presence of cystic duct remnant.* This examination has also been advocated as a method of differentiating acute cholecystitis from acute pancreatitis, which is a frequent and important clinical problem. It may often be difficult to distinguish the two diseases either by symptomatology or clinical findings, and frequently both have histories of previous colic. Keeping in mind

the commonly accepted pathogenesis of acute cholecystitis, that is, cystic duct obstruction, one should be able to opacify the gallbladder in the patient with acute pancreatitis, whereas the gallbladder cannot fill because of the obstructed duct in the patient with acute cholecystitis. Therefore, if an intravenous cholangiogram is done, and this can be done independent of gastric intolerance or faulty absorption and in a fairly short interval of time, the presence or absence of gallbladder filling will assist one in making the differentiation.

Oral cholangiography is valuable in certain cases, particularly when there is some contraindication to the use of intravenous material. In some cases, we seem to have obtained a better concentration by the oral method than by the intravenous one. We have used the usual method of giving the patient 18 Telepaque tablets in divided doses in twelve hours. Films are then made in the usual positions for hepatic duct and gallbladder study.

Operative and postoperative cholangiography is usually done through a T tube. Operative cholangiography requires close cooperation between surgeon and radiologist and is certainly not an examination which can be used only occasionally; it should be used almost as an operative routine, if at all. If employed only occasionally, it is subject to many errors. Cholangiography through the T tube entails certain risks which are not always appreciated. If too great a pressure is applied during the injection, cholangitis, bacteremia, liver abscess, and pancreatitis may result. It is generally advisable to use little, if any pressure, during the injection. In our radiology department, we ordinarily utilize only the pressure of gravity to inject the contrast through the T tube.

GALLBLADDER

In the routine examination of the gallbladder, a plain, predye 14 x 17 scout film of the abdomen is of considerable importance but frequently is not done. On such a plain film, one may often detect gallbladder calculi, a calcified gallbladder, air in the biliary ducts, or other abdominal lesions. However, the routine gallbladder study usually refers to the use of oral contrast material, such as Telepaque, given the evening before. In nearly all cases, 2 or 3 gm. of Telepaque is adequate. One begins with a right upper quadrant film to localize the gallbladder and follows this by more detailed studies, unless obvious calculi are detected at once, in which case the examination can be discontinued. "Cone down" films are then made over the gallbladder, and

these are followed by a lateral decubitus film, a very important part of the examination. The film can be made with equipment present in most x-ray departments. The patient lies on his right side, and the x-ray beam passes horizontally through the gallbladder area. In our department, we make this film with the patient lying on a stretcher, the film in the Bucky of the x-ray table in the upright position. This allows separation of the gallbladder from gas in the bowel, and calculi are often visualized which could not be seen on any other film.

Fluoroscopy of the gallbladder, to position it away from bowel shadows, followed by spot films may be valuable. A postfat meal contraction study occasionally allows detection of small stones in the smaller volume of contrast material that is present after the contraction. Whether or not the gallbladder contracts has not been considered a significant clinical finding.

Besides the usual preparation of the patient for this examination, a fatty meal given at noon the day preceding the examination to empty the gallbladder has been of some value, because then one is more confident that the gallbladder had space available to receive the dye.

It should be emphasized that the routine oral gallbladder examination deserves as much individualization as any other examination. One tends to look upon a gallbladder study as a simple procedure and may become careless in interpretation. Consultation between the clinician and radiologist is often of considerable value and should always be carried out in any questionable case.

Another method that we have used recently is the four-day Telepaque test. Some gallstones become visible by precipitation of contrast material on their surfaces after prolonged exposure to the contrast material. This is true, particularly, in the case of biliverdin pigment stones. According to Salzman, this is a good method of identifying bile duct stones "which opacify in over 75 per cent of cases. This is probably the best available method of demonstrating bile duct stones in the presence of jaundice." To produce this prolonged exposure to contrast material, the patient is given

1 gm. of Telepaque four times a day for four days. Following this, films are made.

The gallbladder may also be examined by use of intravenous contrast material. Examination is carried out as for intravenous cholangiogram. The dye is concentrated by the liver and the gallbladder fills through the cystic duct. This occurs, as mentioned previously, as long as the cystic duct is patent. The size and shape of the gallbladder and its contents can then be evaluated. This test gives no information in regard to gallbladder function but is valuable in confirming the presence of stones or cystic duct obstruction.

This is probably an appropriate place to put in a word or two about the order in which radiographic examinations are scheduled. We all have seen cases in which several examinations were contemplated and perhaps barium studies were done before gallbladder or intravenous pyelogram studies. This, of course, necessitates a delay to allow the barium to clear from the bowel. One must even think of the possibility of gallbladder examinations interfering with a radioactive iodine uptake test.

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Cirrhosis of the Liver

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THE SUBJECT OF CIRRHOSIS is extensive, and I have therefore concentrated my attention on two life-threatening situations that arise from this disease—hepatic coma and variceal bleeding. I would like to mention a few points about etiology first.

Dr. Gökçen, a resident at the University of Minnesota Hospital, has applied a complement fixation test using normal liver as an antigen against the sera from patients with various types of liver disease. His results on a small series indicate positive complement-fixing antibodies in patients with postnecrotic and biliary cirrhosis, but not with Laennec's cirrhosis, with a clear-cut separation between the two groups. This line of approach is similar to but less clearly defined than that reported by Doniak in Hashimoto's *Thyroiditis*. It suggests that an initial tissue injury of uncertain nature initiates the formation of antibody by the release of a parenchymal substance—in the case of the thyroid, thyroglobulin. An antigen-antibody reaction in the organ then leads to more tissue damage. This is just one new idea in relation to etiology that may bear watching in the future.

Before discussing the important complications, a few points should be mentioned about the basic care of a compensated cirrhotic patient. Diet is important therapeutically only in those cases in which a dietary factor is important in the pathogenesis. This group, except in areas of the world where the diet is markedly deficient, is represented by the alcoholics, who may or may not give a history of dietary insufficiency in addition to their alcoholism. Their liver disease progresses from a fatty state to cirrhosis, and they are the only ones to whom the term Laennec's cirrhosis can be applied. The role of fat infiltration in causing scarring is debatable. That alcohol can initiate this sequence even in the presence of an otherwise adequate diet, possibly because it increases the need for choline, is

probable, but the sequence is more likely in the absence of an adequate diet. All other forms of cirrhosis are nondietary in origin and not only do not respond primarily to dietary management but also progress at various rates independent of treatment. Exceptions are when steroids are beneficial and in the case of hemochromatosis, where phlebotomy is of unquestioned value. Idiopathic, posthepatic, postnecrotic, lipoid, and primary biliary or cholangiolitic cirrroses have less favorable prognoses.

Dietary treatment has included generous protein intake ever since Patek's observation that many patients with cirrhosis tolerate and are undoubtedly benefited by high protein intake. Recent observations have clarified the limitations on protein therapy in those with severe cirrhosis and well-developed portasystemic shunts, both in and around the liver. In milder cases, 80 to 100 gm. of protein is permissible and valuable. The patient must be observed closely for tolerance, however. Protein is a two-edged sword, and the whole care of cirrhosis hinges around this point, which is not widely understood. Protein is important to the regenerating liver, although it is clear that the liver does well for long periods on a very low protein diet. Protein intake probably never needs to be more than 1 gm. per kilogram. In giving protein, however, one must start cautiously and titrate the patient up to tolerance as judged by the tendency toward the development of encephalopathy.

Carbohydrate is more important and should be administered in doses up to 400 gm. unless the patient is obese. Fat can be allowed liberally for palatability of the diet. There is no evidence that fat is harmful even in acute fatty infiltration of the liver.

If the patient follows the foregoing diet, there is no need for added vitamins, choline, or methionine. If anorexia is pronounced or if the caloric intake is mainly intravenous, vitamins must be added as mixed capsules or intravenously. In these cases, choline may hasten improvement, but methionine has not been shown to be of additional value and may precipitate stupor.

Of course the most important dietary measure

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in the long run is alcoholic abstinence, because, although minor drinking in the face of a good diet is probably not harmful, people with Laennec's cirrhosis can rarely draw the line. Considerations along the line of promoting abstinence, although probably more important than the things we shall discuss, are beyond the scope of this paper.

Even in the absence of edema or ascites, it is probably wise to restrict salt to 2 to 4 gm. except in hot weather. Substitutes containing potassium glutamate and potassium chloride are the most useful, but their use demands a proof of good renal function.

Adrenal steroid therapy should be reserved for the case of a hospitalized patient in whom marked anorexia and jaundice have persisted despite ordinary supportive measures, especially if accompanied by evidence of increased hemolytic activity. The usual dangers in steroid, bleeding and fluid collection, are particularly dangerous in the cirrhotic patient. Prednisone, prednisolone, and triamcinolone still have a strong tendency to stimulate gastroduodenal ulceration, although the sodium retention is diminished. How much variceal bleeding is due to peptic activity is not known, but certainly the patient on steroids should be protected by antacids and anticholinergics. Steroids have their greatest usefulness in inducing remissions in the course of idiopathic, posthepatic, or lupoid cirrhoses—those of the nondietary type.

It is interesting that the most marked remissions are seen in patients with the greatest elevations of globulins. If we revert to Dr. Gökce's work, one might speculate that hyperglobulinemia is a reflection of the antibody content of the serum and that steroid therapy interferes with this phenomenon, as it does in other antigen-antibody systems, and may depress the formation of antibodies. Unfortunately, these cases frequently exacerbate if the steroid is stopped or decreased. Administration of steroids nearly always results in some diminution of jaundice. It is well to note that this is seen both in the intrahepatic obstruction of cholangiolitic and primary biliary cirrhoses and in extrahepatic obstruction, so that this is no differential test of any reliability.

ASCITES

The special problem of ascites needs some attention. It used to be felt that ascites of any appreciable quantity demanded serial paracentesis as long as the patient survived. However, the incidence of peritonitis, hemorrhage, and, particularly, hepatic coma following the removal

of a large amount of fluid has made such removal advisable only if the quantity is sufficient to cause difficulty in breathing and eating.

The triggering of coma by paracentesis is particularly important. It probably results from hypotension and hypovolemia and their effect on liver and brain circulation. Hyponatremia undoubtedly plays a role. Also, there is a tremendous loss of albumin in the fluid, which the liver manufactures only with great difficulty. If orthopnea demands paracentesis, the amount removed should be just enough to restore comfort. Blood pressure should be monitored carefully, and perhaps 1½ liter of 10 per cent dextrose should be infused intravenously during and after the tap.

The value of sodium restriction has been fully established and may have to be reduced to the 200 mg. level. This lowest level of intake is practicable at home only with great difficulty. If the ascites can be controlled at this level of sodium intake with or without diuretics, it may become possible to increase the sodium after several months or a year, depending on the weight of the individual. For a patient who has had ascites, 1 or 2 gm. of salt per day is probably the maximum allowable amount.

Diuretics may be effective in moving ascites. If mercurials alone fail, sometimes aminophylline potentiates their action. Ammonium salts probably never should be used because they readily produce hepatic coma. Diamox has developed a bad reputation for the same reason, but some patients may tolerate it well. Chlorothiazide and hydrochlorothiazide are very effective in treating ascites, as they are in treating other forms of edema. However, both these agents are so potent that they must be used only when the patient is under close observation. Many patients have gone into coma after taking chlorothiazide. We have become convinced that this is due not to toxicity of the drug but to its great potency in removing sodium, chloride, and, especially, potassium from the body. The diuresis of potassium is apparently a variable thing and may be very marked. All of the adverse effects we have seen have been accompanied by hypokalemic alkalosis, and we have found it necessary to supplement potassium chloride in doses of 3 to 6 gm. per day orally in essentially every patient receiving chlorothiazide. Therefore, when it is used in ascites, the danger lies in marked potassium clearance, and supplemental potassium should be given. Probably the most sensitive test is the serum CO₂ combining power, since this may begin to rise before the potassium level has fallen out of the normal

range and shows a beginning alkalosis. Changes of potassium even within the normal range should be watched, because by the time the level gets down to 3.5 mg. per liter, the body is rather depleted. Burchell has shown that the electrocardiogram is sensitive in detecting hypokalemic alkalosis. Of these tests, however, the CO_2 is cheapest, most readily available, and very sensitive, provided one has a baseline value or can rule out other metabolic or respiratory causes for abnormalities.

Salt-poor serum albumin in intravenous doses of 50 gm. for at least five days has been helpful in moving the ascites in otherwise refractory cases. Unfortunately, the albumin is so rapidly lost into the urine, the interstitial fluid, and the ascites fluid itself that the osmotic pressure of the blood is frequently not sufficiently raised for an effect. Also, the material is very expensive, and may cause pulmonary edema and pyrogenic reactions. These problems make its use valuable only in cases otherwise refractory.

Adrenalectomy has been done for ascites to remove the source of excess aldosterone, and the effect was dramatic in at least one patient. But these people are poor surgical candidates, and this procedure hasn't been widely explored. Incidentally, the high levels of aldosterone in cirrhotic patients with ascites may be part of the reason for their chlorothiazide sensitivity, since aldosterone itself favors a hypokalemic alkalosis. With the promised early availability of aldosterone antagonists, the treatment of ascites may become more effective.

Steroids, both alone and in conjunction with other diuretics, have been dramatically effective in some cases of ascites refractory to other measures. Probably less than half of the cases respond well, however. The mechanism of action may be a simple increase in glomerular filtration rate, or there may be suppression of aldosterone production.

HEPATIC COMA

Strict attention should be paid to the recognition of the earliest signs of impending hepatic coma, which frequently occurs in the absence of jaundice or other signs of further change in liver function. Usually, the first signs are changes in affect, as noticed by the family. The patient is inattentive, forgetful, euphoric or depressed, quarrelsome, and, later, confused. Most often, the typical "flapping" tremor is seen before more advanced neurologic signs appear. Later, a positive Babinski sign, hyperreflexia, ankle clonus, positive Hoffmann signs, and spasticity become apparent. Still later, the reflexes diminish to

flaccidity. Dr. Watson puts much store by fetor hepaticus as an early sign. It is a musty, aromatic, slightly sweet, pervading odor which is detected at first when one is close to the patient and later permeates the whole room. The use of paraldehyde obscures this sign, of course.

There is firm evidence that bacterial decomposition of protein in the colon is of great importance in causing hepatic encephalopathy. At the earliest sign of cerebral disturbance, all protein ingestion must be stopped and the gut cleaned out of blood or dietary protein and bacterial activity depressed. In the comatose patient, a large tube should be passed to the stomach and the contents removed. If blood is present, a Sengstaken tube should be used in an attempt to stop the bleeding. Probably the most important function of this tube is the cephalad traction on veins at the cardia of the stomach by the gastric balloon. Indeed, this may be sufficient to stop bleeding without inflation of the esophageal portion. The tube is left in place eight to twelve hours after apparent cessation of bleeding and then deflated and left in place a while longer before it is removed. All this time, continuous aspiration of the stomach allows the blood loss to be followed and the gut to be protected from the blood load.

The liver must be protected from anoxia and low perfusion rate, so vigorous transfusion is necessary. The transfused plasma fortunately contains prothrombin, but parenteral vitamin K should also be pushed. However, we are discussing a patient in or near coma, so the next important step is to instill a large dose of magnesium sulfate into the stomach to purge the intestinal blood and protein. This should be followed by 1 to 2 gm. of neomycin given by mouth or tube. One of the things I should like to emphasize strongly is the value that oral neomycin is proving to have in the treatment and prevention of coma. Not only is it helpful in the bleeder or the comatose patient, but particularly in the ambulatory patient with a tendency toward protein sensitivity. The protein content of the diet can frequently be raised to a more normal range when such patients are protected by oral neomycin, thus allowing them to approach the ideal diet more closely. Therefore, in spite of its expense, it is frequently helpful and should be used in doses of 0.5 gm. 4 times a day. Neomycin very rarely promotes the emergence of *Escherichia coli* and staphylococci. Indeed, some patients have tolerated it for a year with no difficulty. Little is absorbed, so drug sensitivity and toxicity are not seen.

The patient in coma probably should receive

systemic tetracycline as well as neomycin in the gut, since it is not uncommon for colonic bacteria to enter the blood stream. This occurs particularly in coma but apparently can also be a precipitating event. In the restless patient, chloral hydrate can be used orally or rectally. Promazine is preferable to chlorpromazine because of a lower incidence of allergic cholestatic jaundice, and it rarely causes hypotension. Paraldehyde must be fresh from small ampules stored in a dark, cool place, because of the tendency to break down to acetaldehyde with a resultant severe acidosis. It should never be used even orally from an openmouthed, multiple-dose bottle. Short-chain, long-acting barbiturates not dependent on hepatic inactivation, such as phenobarbital, may be used with moderate safety. Opiates are contraindicated.

More specific management of the central nervous system derangement demands an understanding of the mechanism of its production. Every such patient has pathways by which blood may carry cerebral intoxicants from the gut past the liver to the brain, avoiding detoxification. In patients with acutely poor hepatocellular function, the shunt is in the liver itself, as seen in fulminant hepatitis or acute alcoholic fatty metamorphosis. In chronic forms, collaterals are developed within the liver around regenerative cellular nodules or outside the liver by development of the splenic-gastric-esophageal route or the umbilical vein route in response to portal vein hypertension. The surgical shunts are the largest ones, and 15 to 20 per cent of patients with portacaval shunts have intermittent stupor following ingestion of meat.

The nature of the intoxicants is speculative. It is clear that urea or ammonium chloride in the gut or veins can cause encephalopathy as well as protein in the gut. Ammonium normally is present in high concentration in portal blood but not in hepatic vein blood. The failing liver may be unable to metabolize not only that ammonium formed by bacterial action on protein but also that formed by the kidney, peripheral tissues, and brain. The correlation of hepatic coma with blood ammonium levels is strong but not complete.

Other toxic substances of intestinal origin that are carried to the brain may be important. These include many pharmacologically active amines which can be produced by bacterial action on protein but which are difficult to measure and therefore haven't been studied. One observation that may explain the sensitivity of cirrhotic patients to hypokalemia, and thus clarify the difficulties in the use of chlorothiazide, is that alko-

losis may allow ammonium to enter the cell more readily. Still another fact is that ammonium is a stimulant to the respiratory center, so that high ammonium levels may cause the development of a respiratory alkalosis. Roberts has commented on the presence of respiratory alkalosis in hepatic coma. An obvious vicious cycle is possible.

Certainly cerebral function is also dependent on substances synthesized by the liver, such as nucleosides, cytidin, and uridin. These and other substances are undoubtedly deficient in hepatic failure. Bessman has suggested that 5-hydroxytryptophan, the precursor of serotonin, may be deficient, and he showed some improvement in electroencephalograms when it was injected.

The mainstay of coma treatment is simple glucose therapy. Jones noted a 30 per cent fall in the mortality of acute hepatic insufficiency when intensive glucose therapy was given. At least 1,600 calories should be supplied per day as glucose drinks or 20 per cent glucose through intragastric drip. Dextrose, 20 to 40 per cent, can be given in a polyethylene catheter passed through a vein to the cavae, and even this strength solution is tolerated in the large channel. A No. 15 needle in the femoral vein will allow a rather large catheter to be threaded in blindly. Then a twenty-four-hour drip can be maintained for days.

Glutamic acid combines with ammonium to form glutamine. The good results of Walshe and Priest using glutamate therapy in 1953 have not been confirmed under wider application by Alexander, Schwartz, and Sherlock. The compiled results are not convincing enough to recommend its routine use. Infusion of large amounts of glutamine as the sodium salt may promote pulmonary edema. However, in the recent panel on liver disease under the auspices of the American Medical Association, the consensus was that glutamate and arginine have a definite place in the treatment of coma. That place clearly is in those persons with chronic cirrhosis with well-established shunts who are stable until some event precipitates coma, such as a protein load. These agents have little place in the treatment of severe acute hepatitis or acute fatty degeneration. Certainly arginine requires arginase for its action, and this enzyme is located only in functioning liver cells, which are lacking in acute liver disease.

The prognosis in coma depends on the extent of liver cell damage. The patient with chronic cirrhosis who is in coma because of extensive collaterals and high intestinal nitrogen has a fair

prognosis. Acute hepatitis has the worst prognosis, even though steroids may be used with some benefit. In cirrhosis, the outlook is worse in those with ascites, jaundice, or a low serum albumin, all of which are indicative of poor cell function. In all cases, early treatment of precoma is very important to longevity. Prognosis is obviously better for those with a clear precipitating cause, for this allows a positive line of therapy.

VARICEAL BLEEDING

Bleeding varices are the most threatening single complication of cirrhosis. There are threats of exsanguination, massive protein load, and hepatic circulatory embarrassment. Thus, one of the major precipitations of coma is bleeding. Some steps in the care of bleeding varices have been mentioned: namely, intubation with Sengstaken tube, aspiration of blood, instillation of saline purge and neomycin, use of blood (preferably fresh), and administration of vitamin K. Intravenous Premarin in a dose of 20 mg. has been effective in a number of cases and should be given. Pitressin has been shown to lower the portal pressure. Twenty units of Pituitrin or Pitressin is diluted in 200 cc. of 5 per cent dextrose and given intravenously over a period of twenty to thirty minutes or slower if the blood pressure rises appreciably. A side effect of this therapy may be abdominal cramps. The treatment may be repeated at four-hour intervals.

Operative management can take several forms—variceal ligation, esophagogastrectomy, shunt procedures, and gastrectomy. In an emergency, Linton has championed the ligation of varices. This has proved difficult in these circumstances and is not definitive. There has been a greater tendency to use portacaval shunts. The portacaval shunt is more satisfactory than the spleno-renal because there is a greater fall in portal pressure and reclosure is less likely to occur. The spleen is not usually removed at this operation because a fall in portal pressure often reduces

the degree of hypersplenism that had been manifested by thrombocytopenia, leukopenia, or hemolytic anemia. Sometimes this reversal is slow or absent and splenectomy with or without spleno-renal shunt is indicated later in those with severe, persisting hypersplenism.

The selection of cases for a shunt is important, the mortality being 15 to 20 per cent even in selected series. Jaundice, hypoalbuminemia, hypoprothrombinemia, or encephalopathy makes temporization for a moderate period advisable in hopes that some improvement can be obtained by other means first. This may be striking in dietary cirrhosis, and varices may even regress. Numerous cases with proved varices have survived many years.

The question of whether to do shunt procedures before any bleeding episode occurs is difficult to decide. A recent study from Hines Veterans Administration Hospital by Bake, Smith, and Suberman followed 115 cirrhotic patients with varices that had not bled. They were followed from one to six years, with an average of 3.3 years. Of 115 patients, 41 were alive and 74 had died; 28.6 per cent had bled. Of the 74 deaths, 17.3 per cent were due to bleeding, 26 per cent were due to hepatic failure, and 20 per cent were due to other causes. Eleven, or 9.5 per cent, died of exsanguination during their first episode of bleeding.

The objective of a prophylactic shunt is to prevent death during the first period of bleeding. It was estimated that by the time all patients were dead, 10 to 15 per cent would have died of their first bleeding. This is the maximum advantage to be gained by prophylactic shunt. The Hines study concluded that at the present time such surgery offers no clear-cut advantage to these patients. If further experience could (1) separate those most likely to bleed, (2) lower the surgical mortality, and (3) devise surgery that would prevent bleeding for a longer period, prophylactic shunts might be more clearly indicated.

Jaundice

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JAUNDICE refers to the yellow discoloration of the skin and sclerae resulting from accumulation of bile pigments in the blood. The condition usually is apparent clinically at serum bilirubin concentrations of 2 mg. per 100 cc. or more, and the various types of jaundice reflect different disorders of bilirubin metabolism.

NORMAL BILIRUBIN METABOLISM

Bilirubin is derived principally from the breakdown of hemoglobin from erythrocytes in the reticuloendothelial system, but a small proportion—less than 10 per cent—may come from other porphyrins. The pigment, “indirect-reacting” bilirubin, is transported in the blood stream in loose combination with the serum proteins, particularly with the albumin fraction. In the liver, the alcohol-soluble bilirubin is transformed into water-soluble compounds, “direct-reacting” bilirubin. At least 80 per cent is conjugated to monoglucuronide and diglucuronide by the enzymatic action of glucuronyl transferase, and, under normal circumstances, 75 per cent of the pigment is in the diglucuronide form.¹ Bilirubin sulfate and other compounds account for the remaining 20 per cent of the total. Bilirubin glucuronides are excreted in the bile and thus reach the gastrointestinal tract, where they are converted to stercobilin by the action of the gut flora. Approximately 300 mg. of stercobilin is present in the stool every day, the greater proportion of pigment having been reabsorbed into the portal circulation for further metabolism to glucuronides in the liver. A small amount—less than 4 mg. per day—is excreted in the urine as urobilin.

DERANGED BILIRUBIN METABOLISM

Three groups of pathologic conditions are associated with derangement of bilirubin metabolism leading to jaundice.

1. The increase in breakdown of red cells that

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occurs in various hemolytic states results in an increased amount of indirect-reacting bilirubin in the blood, hemolytic jaundice. Consequently, an increased amount of bile is secreted by the liver and excess fecal stercobilin and urinary urobilin are found.

2. Liver cell disease, hepatocellular jaundice, impairs both the conjugation of bilirubin to glucuronides and the subsequent excretion of these substances in the bile. Bile pigment and excess urobilin appear in the urine, since inefficient hepatic metabolism of glucuronide results in its absorption into the systemic blood stream and excretion by the kidneys, while the utilization of stercobilin from the intestines is similarly impaired.

3. Intrahepatic or extrahepatic biliary obstruction, “obstructive” jaundice, prevents normally conjugated bilirubin from reaching the intestines. Therefore, the stools are acholic and urobilin correspondingly is absent from the urine, and the reabsorption of water-soluble conjugated bilirubin into the blood stream leads to the appearance of jaundice with bile in the urine.

The hitherto unknown mechanism of the van den Bergh reaction can be explained through the recent recognition of glucuronide formation as the key process in metabolism of bile pigment by the liver. Bilirubin itself, being soluble in alcohol but not in water, gives the indirect reaction and does not appear in the urine. In contrast, the glucuronides are water-soluble and, therefore, can give a direct van den Bergh reaction and enter the urine. The renal threshold for bilirubin glucuronide varies around a mean concentration of 3 mg. per 100 cc. of serum.

SOME CLINICAL ASPECTS OF JAUNDICE

As jaundice may result from any type of liver cell disease or from obstruction anywhere in the biliary system, only those aspects of special relevance will be reviewed. Particular reference will be made to recent advances in diagnosis and treatment. There is much current interest in an uncommon group of conditions known as idiopathic hyperbilirubinemias. Usually, but not invariably, these are benign disorders in which

jaundice without other specific symptoms recurs throughout life in patients who frequently reveal a familial history of the disease. The best documented type is that described by Gilbert and Lereboullet² and known as constitutional hepatic dysfunction. Elevation of serum (indirect) bilirubin occurs in the absence of excessive hemolysis or demonstrable liver disease.³ Deficiency of the enzyme glucuronyl transferase has been suggested to account for the condition. A similar but more severe disorder occurring in young children has been described by Crigler and Najjar.⁴ Again, the bilirubin is in the unconjugated form and a transferase deficiency is postulated. Moreover, in this latter condition, deep jaundice may occur; and, as indirect bilirubin is soluble in fat as well as alcohol, damage to the central nervous system leads to neurologic disorder and death.

In contrast, hyperbilirubinemias associated with conjugated bilirubin and bile in the urine have also been described. In the Dubin-Johnson type,⁵ the diagnostic feature is the presence in the liver cells of a pigment thought to be a lipofuscin. Associated abnormalities often are found on further investigation, and other related disorders have been documented in which the pigment is absent.⁶ The mechanism involved is unknown, but it presumably affects the excretion rather than conjugation of bilirubin in the liver.

Another instance in which the differential diagnosis of jaundice may be particularly difficult is the neonatal period. Severe hemolytic jaundice caused by Rhesus incompatibility and leading to neurologic complications due to the solubility of indirect bilirubin in the fat of the central nervous system (kernicterus) may necessitate exchange transfusions when serum bilirubin levels rise above 15 mg. per 100 cc. Large amounts of circulating indirect bilirubin may accumulate also in premature infants, sometimes causing the same complications and requiring the same treatment. This so-called physiologic jaundice once was thought to be due to excessive destruction of fetal red cells, but it is now attributed to a lack of glucuronyl transferase in the immature liver and a consequent inability to conjugate bilirubin.

Various infections of the liver may give rise to hepatocellular jaundice in the neonatal period, and it is increasingly obvious that viral hepatitis can occur at this time. It is postulated that the mother is a carrier of the serum hepatitis virus, although she may give no history of an overt attack, and successive children may be affected. This jaundice may be difficult to differentiate from obstructive jaundice due to bil-

iary atresia, since liver function tests are less reliable in the newborn for reasons including the infant's inability to produce globulins.

DIFFERENTIAL DIAGNOSIS OF JAUNDICE

In the great majority of jaundice cases, a fairly confident diagnosis can be made on the bases of a careful history and physical examination. Apart from the presenting features, effort should be made to elicit details, such as recent injections, contact with hepatitis or tooth extractions, consumption of drugs or alcohol, and a previous or familial history of jaundice. Physical examination should not overlook the vascular, endocrine, nutritional, or cutaneous changes often associated with liver cell disease; the findings of splenomegaly, superficial collateral veins, or a venous hum indicating portal hypertension; and the bruising, scratch marks, and xanthomata sometimes complicating obstructive jaundice.

When difficulty exists, the diagnosis often can be clarified by the use of certain biochemical investigations known, sometimes incorrectly, as liver function tests. Depression of the serum albumin concentration commonly occurs in hepatocellular jaundice, often as a result of impaired synthesis of protein by the diseased liver but sometimes as a reflection of primary malnutrition, as in chronic alcoholism, or dilution of plasma constituents due to the accumulation of extracellular fluid, especially in patients with ascites. The concentration of serum globulin may be elevated in both obstructive and hepatocellular jaundice. In the former, the alpha-2-beta-globulins are moderately increased, whereas a high gamma-globulin peak occurs with liver cell disease. Various nonspecific flocculation tests, such as the thymol turbidity, cephalin cholesterol, and zinc sulfate reactions, reflect some of these changes in the serum proteins and often give a positive result in hepatocellular jaundice due to liver disease. The concentration of serum alkaline phosphatase may be increased in both hepatocellular and obstructive jaundice, but levels of more than twice normal usually indicate obstruction. In both conditions, the prothrombin time occasionally is prolonged, but it is more likely to respond to vitamin K therapy when jaundice is obstructive, as under that circumstance the jaundice reflects impaired absorption of vitamin K due to the steatorrhea rather than inability of the diseased liver to synthesize prothrombin. Unfortunately, the bromsulphalein clearance test is invalidated in jaundice, but it may be helpful as an index of cell function when very small elevations of serum bilirubin concentration occur in liver cell disease.

More recently, the liberation of transaminases from damaged cells into the blood stream has been exploited as a useful liver function test.⁷ Elevated blood enzyme levels occur in diseases of various organs, and the serum glutamic-pyruvic transaminase (GPT) concentration is the most specific for liver cell injury. As with other tests, there is some overlap between patients with liver cell disease and those with obstructive jaundice. However, the finding of a very high level, more than 10 times normal, indicates viral hepatitis or active cirrhosis.

In patients with hepatitis or other disease of the liver cells, the serum transaminase concentrations do not correlate well with serum bilirubin levels—a fact suggesting that the total serum bilirubin concentration reflects factors other than, or in addition to, those directly concerned with liver function. Probably the degree of conjugation of bilirubin is a more sensitive indication of liver cell damage in hepatocellular jaundice.

The indirect fraction of serum bilirubin commonly is elevated in patients with liver disease, but most of the pigment is in the glucuronide form. A smaller proportion appears as diglucuronide than in obstruction, however, and the presence of large amounts of monoglucuronide is an unfavorable finding.⁸ Thus, parenchymal liver disease affects bilirubin metabolism partly by impairing the conjugation of indirect bilirubin to bilirubin glucuronides but, more particularly, by reducing the amount of glucuronide conjugated to diglucuronide. As large amounts of both monoglucuronide and diglucuronide may accumulate in the serum of such patients, defective excretion of these pigments resulting from either enzymatic or mechanical changes due to liver cell damage must also be postulated.

OBSTRUCTIVE JAUNDICE

In conclusion, that which is perhaps the most common cause of difficulty in the diagnosis of jaundice should be considered, namely, the accurate distinction in some cases between so-called medical and surgical jaundices. It is becoming increasingly clear that intrahepatic lesions due to viral hepatitis or drugs, such as chlorpromazine, may present features which are clinically, biochemically, and histologically suggestive of obstruction and which cannot always be differentiated from the features of extrahepatic obstruction by tumors, gallstones, or strictures. It must be emphasized that the history and associated findings usually indicate the diagnosis fairly definitely, but sometimes these are

nonspecific. The clinical features are those of relative well-being, slowly progressive jaundice, and itching of the skin. Hepatomegaly occurs in most patients with obstructive jaundice, regardless of its cause; but the finding of an enlarged gallbladder may be helpful, as it suggests an extrahepatic lesion.

In long-continued obstructive jaundice, nutritional complications may be caused by the steatorrhea, and liver cell failure ultimately can develop from secondary biliary cirrhosis. The bruising and hemorrhages due to impaired absorption of vitamin K, together with skeletal changes caused by malabsorption of vitamin D, calcium, or protein (manifested by osteomalacia or osteoporosis), may be prevented by weekly injections of vitamins A, D, and K accompanied by an ample food intake. Itching usually responds to methyltestosterone tablets, 10 mg. given sublingually once or twice daily, and good results from the use of trimeprazine (Temaril) have been reported also.

Additional diagnostic tests in such cases include examination of the stools for occult blood, since many tumors of the ampullary region or bile ducts are associated with gastrointestinal bleeding. A roentgenogram of the abdomen should be studied for evidence of gallstones, even though only 10 per cent of them are radiopaque, but other radiologic examinations of the biliary system are invalidated by jaundice. Roentgenograms of the stomach and duodenum sometimes show deformity of the duodenal loop due to a neoplasm in the head of the pancreas; and duodenal drainage has its advocates, the most significant result of this time-consuming procedure being the aspiration of blood-stained material.

Should the diagnosis remain in doubt, liver biopsy can be performed. This investigation is contraindicated by hematologic abnormalities, such as a prolonged prothrombin time; but the presence of jaundice does not preclude it, and often it is preferable to laparotomy. Unfortunately, the histologic findings frequently fail to differentiate convincingly between intrahepatic and extrahepatic obstruction.

The administration of 60 mits of corticotropin daily or 20 mg. of prednisone three times a day for five days may be useful in diagnosis.^{9,10} If there is a 50 per cent reduction in the serum bilirubin level within this time, treatment should be continued. A progressive fall to normal levels confirms the diagnosis of hepatitis. Unfortunately, this test does not give a definite result in every case, since the effect of these drugs on jaundice is somewhat nonspecific and not every

patient with hepatitis responds dramatically. Nevertheless, it can eliminate some patients from the list of potential surgical candidates.

Surgical intervention is contraindicated when the diagnosis is seriously in doubt, as fatal hepatic coma following laparotomy for suspected biliary obstruction is one of the most common causes of death in viral hepatitis. Moreover, in malignancy involving the head of the pancreas, ampulla, and bile ducts, the collective surgical results are not sufficiently good to make immediate operation imperative. As obstructive jaundice is harmless over periods of a few weeks, providing that the nutritional therapy previously outlined is implemented, it is justifiable to delay operation until additional information, especially the effect of time, can be acquired and assessed. Nevertheless, a small group will remain in whom surgery is required for diagnosis. Inevitably, this group will still include the occasional patient in whom hepatitis is found later.

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THE STEROIDAL pattern of primary aldosteronism can be classified according to potassium loss, arterial hypertension, hyperaldosteronuria, and adrenal anatomic abnormality.

Nomenclature varies with conditions, the following names having been suggested: (1) primary hyperaldosteronism when urinary 17-ketosteroids and 17-hydroxysteroids are normal; (2) pure primary hyperaldosteronism when corticosterone is normal; (3) primary hyperaldosteronism with hypercorticosteronism when corticosterone is elevated; and (4) mixed hypercorticism including hyperaldosteronism when 17-ketosteroids, 17-hydroxysteroids, 17-ketogenic steroids, or other steroids are high.

In a typical case of pure primary hyperaldosteronism, analysis of urine for known steroids revealed no significant abnormalities other than hyperaldosteronuria and low sodium excretion. Secretions of an adenoma found at surgery seemed to be affected by hormonal and humoral factors. It is thought that the adenoma develops by way of a nontumoral hyperplastic state.

An approximate parallelism between sodium and potassium balances was found upon metabolic examination. A lack of correlation between aldosteronuria and sodium balance was also noted.

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Problems of Gallbladder Disease

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OUR ATTITUDE toward the management of acute or chronic gallbladder disease must be based on the fact that the initial complications are due to acute, recurrent, or chronic cystic duct obstruction. This is usually caused by a stone with its attendant edema, congestion, and varying degrees of infection of the gallbladder wall. The final pathologic process varies within wide limits. The cystic duct obstruction may be painless, as in chronic hydrops, or excruciatingly painful, as in acute empyema.

The patient with gallstone disease is fortunate indeed if he does not fall victim to the extremes of medical and surgical opinion. *These extreme views may be stated as: no operation for stones except for biliary colic or, conversely, immediate cholecystectomy for gallstones "on discovery."*

1. Cholecystography

In the diagnosis of gallstone disease the most important evidence is the demonstration of positive or negative shadows on the x-ray film after the administration of Teridax or Telepaque. Reliance is also placed on nonvisualization after administration of the dye on two successive days. If clinical suspicion of gallstones is high, I tell the patient that I am not disturbed that the radiologist fails occasionally to demonstrate a lesion but rather that I am amazed at how frequently he does provide the final answer. This approach immediately focuses the patient's mind on the most important single factor in the diagnosis of gastrointestinal disease—a careful recheck of the clinical history. A similar attitude is also necessary when stones are found. The clinician must not too readily ascribe all of the patient's symptoms to the gallstones.

The presence of gallstones does not always justify a final diagnosis or an abdominal operation. Their demonstration often represents the beginning and not the end of the investigation.

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Even a greater need for reassessment of the patient's symptoms before surgery is indicated in patients with nonvisualizing gallbladders. Inquiry into causes outside the biliary tract for nonvisualization must be made.

2. Cholangiography

For our purposes, we may subdivide cholangiography into (a) indirect and (b) direct, at or after operation.

Indirect cholangiography is in a transitional stage of development and is still a crude screening method.

Operative cholangiography can be satisfactory, provided that the same quality of x-ray equipment is available in the operating theatre as in the department of radiology.

Direct postoperative cholangiography is a reasonably precise method of demonstrating calculi and strictures.

3. The interpretation of symptoms and signs

The surgeon should not advise immediate cholecystectomy for gallstones associated with painless, flatulent indigestion without first considering other possible explanations for the patient's complaints.

With regard to pain, one proven attack of biliary colic is all the warning needed to justify cholecystectomy. Pain may be atypical both as regards location and reference.

4. Complications of delay in treatment

In any case in which gallstones have been demonstrated, the possibility of complications must not be ignored.

Young patients with multiple, silent, small stones should undergo cholecystectomy because risk of future complications is higher than the risk of elective surgery. A patient with a large, solitary, cholesterol stone—in a gallbladder otherwise normal on inspection—may have the stone removed and the gallbladder drained.

Serious complications to be prevented include perforation of the gallbladder with peritonitis or abscess, common duct stone, jaundice, suppurative cholangitis, secondary biliary cirrhosis, pancreatitis, carcinoma of the gallbladder, duodenal fistula, and gallstone ileus.

5. Associated disease

Patients with myocardial disease may benefit by a cholecystectomy if the gallbladder is diseased and causing colic.

Records show that, in diabetes, stones are present in 30 per cent of all diabetics over 20 years of age and in 1 out of 2 over 50. Acute pancreatitis is associated with gallstones in 75 per cent of cases. In carcinoma of the gallbladder, over 90 per cent of patients have stones.

6. The jaundiced patient

The jaundiced patient presents an interesting problem. Seventy-five per cent can be diagnosed on clinical evidence alone and 90 per cent after combined clinical and laboratory investigation. Our first concern is to separate the medical from the surgical case.

The patient who is admitted with recent severe pain and variable, transient jaundice presents little diagnostic difficulty. Operation should be delayed as long as the patient improves.

In persistent and more severe jaundice, the diagnosis of obstruction due to stone or stricture is supported by a history of colic; previous gallbladder surgery; fluctuating jaundice, with or without fever; severe pruritus; absence of grossly enlarged liver; absence of a palpable spleen or ascites; and absence of the more remote signs of intrahepatic disease, such as spiders and palmar erythema.

Cases difficult to distinguish from obstructive jaundice are those due to homologous serum jaundice, viral hepatitis, or drug hepatitis.

Laboratory tests in favor of obstructive jaundice are bile in the urine, stools variable but rarely clay-colored, negative flocculation tests, high alkaline phosphatase (which may, however, be normal in recent jaundice), normal blood proteins, and the quick response to vitamin K if the prothrombin time is abnormal.

Liver biopsy is helpful in ruling out viral hepatitis, drug hepatitis, and intrahepatic secondary malignancy. It is contraindicated and dangerous in cases of prolonged obstructive jaundice in those that do not respond to vitamin K, and in cases with ascites.

When diagnosis is still doubtful after two weeks of investigation, an exploration is indicated, provided the prothrombin time and blood proteins are satisfactory.

7. The treatment of acute cholecystitis

In conservative treatment of acute cholecystitis, emergency surgery is only performed in the

fulminating case, after observation for twenty-four to forty-eight hours, and when there is (1) increasing pain in spite of repeated analgesics, (2) increasing local tenderness and rigidity, or (3) increasing fever and leukocyte count. Some surgeons advocate early operation in all cases.

Whether one favors immediate surgery or prefers to wait, conservatism in the extent of the surgical procedure is the first rule. Surgery should be confined to the gallbladder itself. The common duct should not be explored during the acute phase. However, a "safety valve" procedure in emergency cholecystectomy has been practiced by me for many years. After the gallbladder has been freed and removed, a fine catheter is led through the stump of the cystic duct into the common bile duct. This catheter is sutured to the duct with fine chromic gut and brought out through the wound together with a Penrose drain. The maneuver reduces the danger of bile leakage if there is distal obstruction of the common bile duct.

On occasion, the gallbladder and surrounding structures become so friable that the cystic duct and even the cystic artery may be severed before formal ligation can take place. It is dangerous to clamp blindly with a forceps for these structures. A firm pack inserted into the region and left for five minutes usually controls the bleeding, and the artery and duct may be identified and clamped in a bloodless field.

Another maneuver is to have the assistant compress the structures of the right border of the gastrohepatic ligament between the index finger and thumb of his left hand. This effectively controls the hepatic artery so that the blood can be aspirated from the surgical field and the structures visualized clearly. When finger pressure is momentarily released, a small spurt of blood readily identifies the cystic artery so that it may be clamped and tied under direct vision.

8. Exploration of common duct

There are at least 7 indications for exploration of the common bile ducts: (1) jaundice, (2) stones palpable in the common bile duct, (3) dilated duct, (4) thickened common bile duct, (5) flecked bile on aspiration of common bile duct, (6) induration of head of pancreas, and (7) multiple small stones in gallbladder.

9. Pain and/or jaundice after cholecystectomy

This may be due to (1) a stone left in the common duct, (2) unrecognized peptic ulcer, (3) subacute or chronic pancreatitis, (4) cystic duct remnant containing stones, or (5) stricture.

MEDICAL GRAND ROUNDS

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Cardiovascular Complications of Myxedema

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THE PURPOSE of this paper is to discuss the cardiac diseases associated with myxedema—myxedema heart and arteriosclerotic heart disease. A series of myxedematous patients at the Minneapolis Veterans Hospital was analyzed to determine the incidence of cardiac complications.

MYXEDEMA HEART

Myxedema heart was first described by Zondek in 1918.¹ He characterized it as follows:

Before treatment with thyroid: (1) enlargement of both sides of the heart, (2) slow, indolent heart action with normal blood pressure, and (3) lowering of P and T waves on the electrocardiogram. After treatment with thyroid: (1) return of the heart to near normal size, (2) more lively heart action, more rapid pulse, and unchanged blood pressure, and (3) gradual return of P and T waves to normal.

Myxedema heart was first described in the United States by Fahr² in 1925. He described the cases of 3 patients with this condition who showed dyspnea, rales at the lung bases, ascites, enlargement of the liver, and pitting edema. The cardiac shadow was enlarged in a characteristic manner, suggesting dilation of all chambers. Electrocardiograms showed absent or negative T waves in lead I and negative QRS in lead III. These cases did not respond to bed rest and digitalis but were "cured" by desiccated thyroid.

In 1932, Fahr³ described 17 cases of myxedema, with symptoms of heart failure in 75 per cent of the patients. All improved on thyroid. One patient was taken off thyroid and observed in the hospital. The basal metabolic rate fell to -30 per cent in ten weeks. Signs of early myx-

edema were noted twelve weeks after thyroid was discontinued. Flattening of the T wave in lead I occurred at ten weeks, but cardiomegaly and symptoms of congestive failure did not develop for eight months.

In 1933, Lerman and associates⁴ studied 30 myxedematous patients with serial chest roentgenograms and electrocardiograms before and during treatment. The heart was usually enlarged before treatment and usually decreased on thyroid therapy, with maximum shrinkage between three and six months. The most common electrocardiographic abnormalities were flat or inverted T waves, low voltage of the QRS, and low P waves. The greatest electrocardiographic improvement was usually associated with the most marked diminution of the heart. Only 4 patients had evidence of congestive heart failure. Of these, 3 had evidence of peripheral arteriosclerosis; all were hypertensive and improved on thyroid.

Clinical findings. The symptoms of myxedema heart, in addition to the usual symptoms of myxedema, are variable. Often only slight dyspnea on exertion and palpitation are noted.⁵ Less commonly, symptoms of severe congestive heart failure are present.³ Myxedema heart is usually found only with well-developed hypothyroidism.

Signs of myxedema heart are (1) Difficulty in perceiving the apex beat, (2) muffling of the cardiac tones, (3) bradycardia, and (4) increase in the size of the heart.⁵

The electrocardiographic abnormalities consist of low voltage of all components, with flattening and inversion of the T waves and reversion toward normal on thyroid. Various degrees of atrioventricular block have been reported.⁶ White showed that the electrocardiographic abnormalities were not caused by the increased resistance of the myxedematous skin.⁴ He ob-

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tained tracings with subcutaneous needle electrodes which showed similar results.

Roentgen study shows increased size of the cardiac shadow to the right and left. At fluoroscopy, decreased amplitude of contractions and bradycardia give the impression of a sluggish heart.

The findings in myxedema heart usually return to normal after treatment with thyroid, though this return may be slow. In one case, the patient became euthyroid, and the electrocardiogram returned to normal only after twenty-nine months of treatment.⁷

Many of the findings in myxedema heart can be explained on the basis of pericardial effusion. Kern and associates⁸ reported 4 consecutive patients with myxedema who had effusions demonstrated by pericardiocentesis. One patient showed elevation of the T waves immediately after the tap, suggesting that this abnormality was caused by the effusion. Cardiac tamponade was not produced, probably because of the great distensibility of the pericardium. Myxedematous patients with pericardial effusions are usually women, 50 to 70 years old, with symptoms of more than three years' duration. Pleural and peritoneal effusions are frequently found in addition to pericardial effusions.⁹

There are well-documented cases of myxedema heart without pericardial effusions.⁷ In a study of 34 cases of myxedema, cardiomegaly was found in 53 per cent. Of 9 pericardial taps, 7 showed fluid, but the amount was usually not enough to account for all of the enlargement of the cardiac shadow. The T waves returned to normal after thyroid treatment but not immediately after the tap.¹⁰

Hemodynamics. The cardiac output in myxedema is reduced. The decrease is usually proportional to the decreased oxygen consumption.¹¹ The arteriovenous oxygen difference is not changed. In a case of myxedema heart, however, the decrease in cardiac output was out of proportion to the decreased oxygen consumption, and the arteriovenous oxygen difference was increased.¹²

The circulation time is prolonged in myxedema.¹³ Peripheral resistance is increased, and blood and plasma volumes are lowered.¹² Blood pressure is variable.¹⁴ Capillary permeability is increased, with return to normal after treatment.¹⁵ Serum sodium concentration tends to be low, and the radioactive sodium space is increased. Both tend to return to normal with treatment. There is little change in exchangeable potassium. These results may be due to the interstitial deposition of osmotically active colloid

substances with migration of fluid into the extracellular spaces.¹⁶

Davies and associates¹⁷ showed that patients with myxedema do not retain salt and water. A group of myxedematous patients were compared with a group having rheumatic heart disease. Cardiac output, glomerular filtration rate, and renal blood flow were all reduced about the same degree in both groups. On a daily intake of 10 gm. of sodium chloride, the rheumatic heart patients retained water and salt and gained weight. The myxedematous patients did not. A patient with combined mitral stenosis and myxedema also did not retain water. The authors suggest that myxedema affects the renal tubular reabsorption of salt and water.

Many of the signs of congestive heart failure may be found in myxedema. However, Friedberg¹⁸ has pointed out several differences between myxedema and heart failure. In contradistinction to heart failure, the edema fluid in myxedema is of high specific gravity, venous pressure is normal, blood volume is decreased, and digitalis evokes no response. Some of the signs in myxedema may be caused by complications, such as serous effusions and anemia, rather than by true congestive heart failure.

Pathology. Because the mortality of myxedema heart is low, the autopsy experience is not great. In a typical case, the heart is globular with dilation of all chambers. The myocardium appears pale and flabby. Microscopic examination of the fibers shows loss of striation, branching, pyknotic nuclei, irregular staining, and vacuoles containing a basophilic material. Similar findings have been observed in beriberi heart disease.^{19,20} Other findings include thickening of the capillary basement membranes,²¹ interstitial edema, and fibrosis.¹⁴

Basophilic degeneration of the myxedema heart has been of considerable interest. It is a nonspecific change and may be seen to a lesser degree in normal hearts. It is often found in association with lipochrome, the "wear-and-tear" pigment.²² Myxedematous skin contains an amorphous material in the corium that is digested by hyaluronidase.²³ The basophilic substance in the heart is periodic-acid-Schiff positive, stains with Best's carmine, and is unaffected by hyaluronidase.²⁴ It has been suggested that this material, a mucoprotein or mucopolysaccharide, increases the extravascular colloid osmotic pressure, resulting in interstitial edema. In other words, the process that causes myxedema of the skin may be similar to the one that affects the heart. This alteration in osmotic balance may also be responsible for the formation of effusions.

The association of myxedema with arteriosclerotic heart disease is well known. Fishberg²⁵ in 1924, reported a case of a 21-year-old man with hypothyroidism who died with severe generalized arteriosclerosis. In a study of 59 patients with myxedema, Bartels and Bell²⁶ found a 25 per cent incidence of coronary arteriosclerosis as shown by angina pectoris or coronary thrombosis. In a series of 18 autopsied cases of myxedema, coronary sclerosis was found in 12.²¹ The high incidence of coronary artery disease is striking, since most myxedema patients are women, who usually have a low incidence of arteriosclerotic heart disease.

Kountz²⁷ found medial cystic degeneration and calcification in the aorta and large arteries of patients with myxedema. He reported 3 hypothyroid patients who had died of rupture of the aorta and 1 who had died of coronary occlusion.

Angina pectoris and myocardial infarction. Angina pectoris may occur in myxedema after the start of thyroid therapy. Sclerotic coronary arteries, able to supply the decreased demands of the myxedematous heart, become relatively insufficient with increased cardiac activity on thyroid treatment.

Angina pectoris occurring with untreated myxedema that subsides on thyroid therapy is rare. Mussio Fournier and associates²⁸ found only 6 cases, including their own, in the literature. The cause of the angina in these cases is unknown. Angina pectoris in untreated myxedema may subside on small doses of thyroid but may reappear when thyroid is increased.¹⁴ Thus, it would appear that there are 3 kinds of angina pectoris occurring with myxedema: (1) angina pectoris caused by coronary artery sclerosis and aggravated by thyroid, (2) angina pectoris associated with myxedema and relieved by thyroid (rare), and (3) mixed forms.²⁸

Similarly, myocardial infarctions are frequent after and rare before the onset of thyroid therapy. In Bartels and Bell's²⁶ series, there were 5 deaths, 4 of which were caused by myocardial infarctions that occurred from three weeks to six months after the start of thyroid treatment.

Cholesterol. The role of cholesterol in the pathogenesis of arteriosclerotic heart disease is disputed, but many investigators feel that the high cholesterol levels of myxedema are related to the high incidence of atherosclerosis. Phospholipids are also elevated; neutral fats are unchanged.²⁹ Following thyroidectomy, the serum cholesterol tends to rise as the basal metabolic rate falls.³⁰

Arteriosclerotic lesions have been produced in rabbits but not in dogs by the use of a high-cholesterol diet. Dogs made hypothyroid by administration of radioactive iodine develop atherosclerotic lesions of the large arteries when fed a high-cholesterol diet. This is associated with very high levels of cholesterol, often more than 1,000 mg. per cent.³¹

The harmful effects of elevated cholesterol are disputed by Blumgart and his group.³² For many years, they have been treating patients with severe angina pectoris or congestive heart failure by producing a hypothyroid state. This was originally done by thyroidectomy and, more recently, by radioactive iodine ablation of the thyroid. In a series of 8 patients who had total thyroidectomy because of rheumatic heart disease or cor pulmonale, the average preoperative cholesterol concentration was 170 mg. per cent and the average rise was 125 mg. per cent. The basal metabolic rate was maintained at about -20 per cent with small doses of thyroid. The patients survived one to thirteen years after thyroidectomy. At autopsy, the incidence of arteriosclerosis was no greater than in euthyroid patients of the same age.

In a series treated with radioactive iodine, the patients were allowed to become markedly hypothyroid. The dose of thyroid was then adjusted to give the most relief with the least amount of hypothyroidism. Usually, 6 to 12 mg. of thyroid per day was used, and the basal metabolic rate was maintained at about -20 per cent. Cholesterol levels became elevated. A few showed increased size of the cardiac silhouette and decreased voltage on the electrocardiogram, but none had increased symptoms of angina pectoris or congestive heart failure.^{33,34} Sitosterols reduced the cholesterol level in 1 patient from 700 to 400 mg. per cent.³⁵ It is possible that the small dose of thyroid which these patients received was sufficient to protect them against the atherosclerogenic effects of myxedema.

Treatment. Because of the danger of precipitating angina pectoris or myocardial infarction, treatment should begin with small doses of thyroid. Angina pectoris has resulted from doses as small as 6 mg. per day.¹⁵ The presence of chest pain in untreated myxedema, however, is not a contraindication to thyroid therapy, since the angina may improve on treatment.

The usual initial dose of thyroid is 4 to 8 mg. per day. This is gradually increased, with the patient under careful observation, until the onset of angina or until the patient becomes euthyroid. The clinical state of the patient, not the basal

TABLE 1

CHARACTERISTICS OF MYXEDEMATOUS PATIENTS WITH ARTERIOSCLEROTIC HEART DISEASE

Case No.	Age	Duration Symptoms years	Cholesterol mg. %	BMR %	Radioactive iodine uptake % 24 hrs.	Protein-bound iodine μ g. %	Hgb. gm. %	EKG		Cardio- megaly	Angina pectoris	Myocardial infarction	Hypertension	Comment
								T waves abnormal	QRS low voltage					
1*	56	7	486	5	10	3.2	14.8	Yes	No	No	Yes	Yes	No	Case reports
2	63	9	325	30	4	1.0	11.8	Yes	Yes	Yes	Yes	No	No	Case reports
3	67	1½	389	21	4	—	12	Yes	Yes	No	Yes	No	Yes	Angina pectoris improved on thyroid treatment
4	55	—	341	—	1.5	—	15	No	No	Yes	Yes	Yes	No	Died of myocardial infarction three years after treatment was begun
5	47	6	428	31	7	1.1	13.5	Yes	No	Yes	Yes	No	No	Angina pectoris developed on thyroid
6	41	5	454	36	3	1.7	14.5	Yes	No	Yes	Yes	Yes	No	Angina pectoris and myocardial infarction on thyroid
7	60	7	329	37	2	—	11.7	Yes	Yes	No	No	Yes	No	Myocardial infarction on treatment
8	66	2	270	39	5	0.9	13.3	Yes	Yes	Yes	No	Yes	No	Myocardial infarction on treatment
9*	37	1	355	30	7	—	14.4	Yes	No	No	Yes	No	Yes	Angina pectoris before treatment; unchanged on treatment
10*	66	½	322	15	9	2.1	14	Yes	No	Yes	No	Yes	No	Myocardial infarction on treatment; arteriosclerotic aneurysm of abdominal aorta
11	68	½	302	25	3	1.2	14.6	Yes	Yes	Yes	Yes	No	No	Heart size returned to normal; developed angina pectoris on treatment.
12	65	¼	311	6	8	2.3	14	No	No	No	Yes	No	No	Angina pectoris improved on treatment
13	65	2	275	35	3	2.4	14	Yes	No	Yes	Yes	Yes	Yes	Old myocardial infarction. Angina unchanged. Arteriosclerotic aneurysm of abdominal aorta
14	49	4	350	12	—	0.6	12.6	No	Yes	No	Yes	No	No	Angina improved on treatment
15	62	2½	316	31	5	—	14.8	Yes	No	No	Yes	Yes	No	Angina increased on treatment; myocardial infarction on treatment
16	63	2	307	17	8	—	12.7	Yes	No	No	Yes	No	No	Angina pectoris on treatment

*Surgical myxedema. All other cases were spontaneous myxedema.
Case 1 was the only woman.

metabolic rate or other laboratory tests, should be the guide to therapy.

CARDIAC COMPLICATIONS OF MYXEDEMA AT MINNEAPOLIS VETERANS HOSPITAL

The charts of all patients with the diagnosis of hypothyroidism seen at the Minneapolis Veterans Hospital since 1952 were reviewed. Only patients with definite clinical and laboratory evidence of myxedema were selected. Patients who had been made hypothyroid in the treatment of severe heart disease were excluded. In 37 patients studied, myxedema was spontaneous in 26, followed surgery in 9, and followed radioactive iodine treatment in 2. All but 1 patient were men.

Arteriosclerotic heart disease, manifested by angina pectoris or myocardial infarction, was present in 16 patients, or 44 per cent. The data concerning these patients are summarized in table 1. Thirteen patients had typical angina pectoris, with chest pain on exertion, which was relieved by rest. Seven had angina before the

start of thyroid therapy. Of these, 3 improved on treatment, 2 were unchanged, and 2 had increased angina. Six patients developed angina during thyroid treatment, but the angina was usually controlled by decreasing the dose of thyroid.

Eight patients had myocardial infarctions; 3 occurred before treatment and 5 during treatment. Arteriosclerotic aneurysms of the abdominal aorta developed in 2 patients. On resection, 1 of these showed medial calcification and cholesterol clefts. Eight patients showed enlarged hearts on chest roentgenograms, and 5 were anemic.

Twenty-one patients did not have angina pectoris or myocardial infarction. Most of these showed low voltage QRS and low or negative T waves on the electrocardiogram. Cardiac enlargement was present in 8 patients but was not typical of myxedema heart and did not return to normal on treatment.

Patients with and without symptomatic arteriosclerotic heart disease were compared with

TABLE 2

COMPARISON OF MYXEDEMATOUS PATIENTS WITH AND WITHOUT ARTERIOSCLEROTIC HEART DISEASE

ASHD		Age	Duration Symptoms Years	BMR %	Radioactive RAIU iodine uptake %	Protein- PBI bound iodine μ g. %	Cholesterol mg. %
	Mean	58	3.1	26	2.7	1.6	340
	Range	37-68	1 ₄ -9	6-39	1.5-9	0.6-2.4	270-486
No ASHD	Mean	46	1.8	22	3.6	1.3	350
	Range	22-78	1 ₁₂ -8	+2-42	0-9	0.6-2.5	133-656

respect to age, duration of symptoms, cholesterol concentration, and thyroid function studies. The results are shown in table 2. Patients with arteriosclerotic manifestations were somewhat older. The difference in average duration of symptoms was not significant ($p > 0.1$). The cholesterol concentrations and thyroid function tests of the two groups were similar.

CASE REPORTS

Case 1. E.H., a 56-year-old white woman who was an employee in the hospital diet kitchen, was admitted to Minneapolis Veterans Hospital in August 1959, complaining of chest pain of twelve hours' duration.

In 1948, she had had a subtotal thyroidectomy for exophthalmic goiter. The pathologic diagnosis was diffuse hyperplasia. In the immediate postoperative period, a psychotic reaction developed, which led to prefrontal lobotomy in 1949.

The patient was first admitted to the Minneapolis Veterans Hospital in May 1953 because of "tightness of the head," which had lasted for one year. She had gained 50 lb. and had had the onset of the menopause following thyroidectomy. Her pulse was 60, and blood pressure was 180/100. She had a full face, dry skin and hair, and slow return of the tendon reflexes. The hemoglobin was 14.7 gm. per cent; cholesterol, 425 mg. per cent; basal metabolic rate, -21 per cent; protein-bound iodine, 2.9 μ g. per cent; and radioiodine uptake, 7.7 per cent in twenty-four hours. A chest roentgenogram

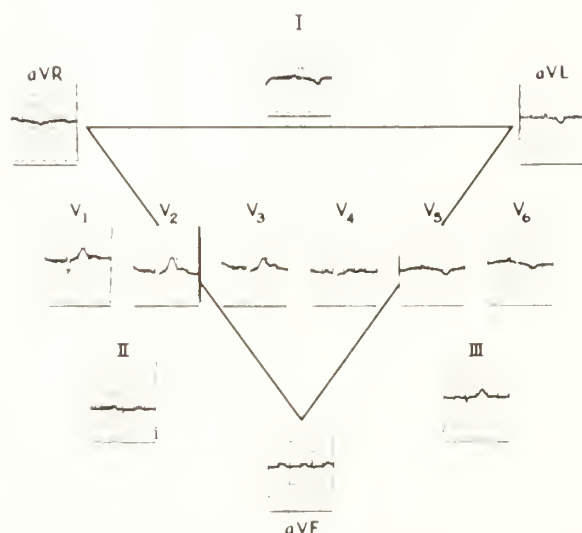


Fig. 1. Case 1. Electrocardiogram in an untreated myxedematous patient after an acute attack of chest pain. The T waves are inverted with coving of the ST segment in leads I, aVL, V₁ and V₂. There is low voltage of the QRS complexes in the limb leads.

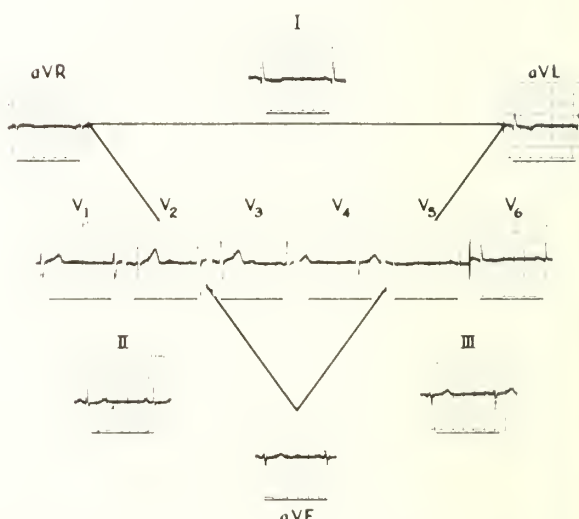


Fig. 2. Case 1. Electrocardiogram two months after the acute attack of chest pain and three weeks after the beginning of thyroid therapy. Q waves have appeared in leads I, aVL, V₁, and V₂. QRS voltage has increased.



Fig. 3. Case 2. Chest roentgenograms in a case of myxedema heart showing decrease in size of cardiac silhouette after five months of thyroid treatment.

was negative and the electrocardiogram showed nonspecific ST changes. She was treated with thyroid, increased from 15 to 90 mg. per day, with noticeable improvement.

The patient was next seen three months later. She had increased the dose of thyroid to 180 mg. per day. Heat intolerance and palpitation were present, but she did not complain of chest pain. The basal metabolic rate was +37 per cent, and the cholesterol was 234 mg. per cent. It was suggested that thyroid be reduced, but, possibly because of a misunderstanding, it was discontinued.

In July 1956, the patient was admitted for the second time for bleeding hemorrhoids. Hemorrhoidectomy was done and a rectal polyp fulgurated.

She was admitted again in August 1958 because of low back pain, urinary and fecal frequency, left lateral chest pain, and fatty food intolerance. Physical examination was unremarkable. Cholesterol was 510 mg. per cent, basal metabolic rate was +10 per cent, and protein-bound iodine was unreliable because of gallbladder dye. A cholecystogram was normal.

In August 1959, the patient was admitted for the fourth time because of progressive dyspnea on exertion and anterior chest pain of two years' duration, which was relieved by rest. On the evening before admission, she had onset of dull, aching chest pain and malaise. On the morning of admission, the pain became very severe, with radiation to the neck and both arms, and dyspnea and sweating occurred.

Physical examination revealed a pale, obese, white woman with puffy face and eyelids. The pulse was 60 and blood pressure was 110/80. There was an old thyroidectomy scar. Skin and hair were dry, with thinning of the lateral portions of the eyebrows. The tendon reflexes showed slow return. The temperature was

96° F. and rose to 100° F. during the first twenty-four hours. The white blood count was 11,500 with 74 per cent neutrophils. Sedimentation rate was 32 mm. Urine showed a trace of albumin. Hemoglobin was 14.8 gm. per cent; blood urea nitrogen, 18 mg. per cent; blood glucose, 93 mg. per cent; cholesterol, 486 mg. per cent; calcium, 9.6 mg. per cent; and phosphorus, 3.4 mg. per cent. Serum glutamic-oxaloacetic transaminase was 190 units. Basal metabolic rate was -5 per cent; protein-bound iodine, 3.2 μ g. per cent; and radioactive iodine uptake, 10 per cent. Chest films and cardiac fluoroscopy were negative. Electrocardiograms showed evidence of anterior and lateral myocardial damage (figures 1 and 2).

The patient was treated with bed rest and anticoagulants. Two weeks after admission, thyroid was begun at 8 mg. per day. Following ambulation, the thyroid was gradually increased. On a dose of 60 mg. of thyroid per day, she complained of chest pain. Subsequently, she was given 45 mg. of thyroid per day. Anticoagulants were continued. Cholesterol fell to 272 mg. per cent. In November 1959, she was again hospitalized with chest pain; electrocardiograms showed further signs of cardiac damage.

Comment. This case illustrates the occurrence of angina pectoris and myocardial infarction in an untreated hypothyroid patient. High cholesterol levels had been present for at least six years.

Case 2. N.W., a 63-year-old white man, was admitted to Minneapolis Veterans Hospital for the first time in September 1954. Since 1945, he had noted weakness, gradual slowing of activity, dyspnea, and intermittent ankle edema. There was no history of chest pain.

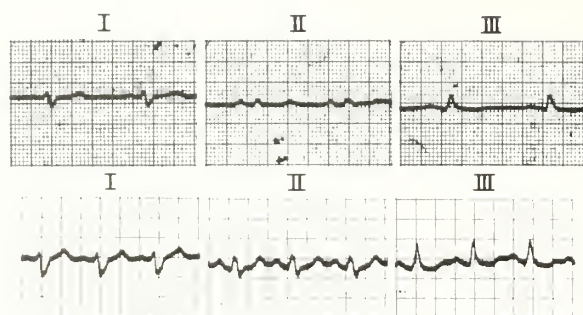


Fig. 4. Case 2. Electrocardiograms in myxedema heart. Before treatment (*above*), there is low voltage of QRS complexes and intraventricular conduction defect. After three months of thyroid therapy, QRS voltage has increased. There is a right bundle-branch block.

The pulse was 72, and blood pressure was 140/90. The skin was dry and scaly with a yellow tinge. The eyelids were puffy, hair was sparse, and voice was coarse. The lungs were clear, the heart was enlarged to percussion, and the heart tones were distant. The liver was not enlarged, and there was no edema. The tendon reflexes showed slow return.

Hemoglobin was 11.8 gm. per cent; cholesterol, 325 mg. per cent; and protein-bound iodine, 1.0 μ g. per cent. Radioactive iodine uptake was 3.9 per cent in twenty-four hours. Following administration of 10 units of thyroid stimulating hormone, the uptake was 4 per cent. Chest roentgenograms and cardiac fluoroscopy showed a generally enlarged cardiac shadow and decreased pulsations (figure 3, *left*). Electrocardiogram showed low voltage of the QRS and intraventricular conduction defect (figure 4).

The patient was treated with thyroid, which was increased from 7.5 to 60 mg. per day. He showed improvement of symptoms and marked decrease in the size of the cardiac shadow (figure 3, *right*). QRS voltage increased, but right bundle-branch block pattern persisted.

Three months later, on a dose of 90 mg. of thyroid, nervousness and angina pectoris had developed. The cholesterol was 135 mg. per cent. Angina improved after the dose of thyroid was reduced to 60 mg. Hemoglobin increased to 13.3 gm. per cent.

Comment. This patient had typical features of myxedema heart. The development of angina pectoris and persistence of abnormal electrocardiogram with treatment indicated that coronary arteriosclerosis was present. Only one other patient had the typical findings of myxedema heart. This patient also had coronary arteriosclerosis, which was demonstrated by calcification of the left coronary artery on fluoroscopy and the development of angina pectoris during thyroid treatment.

SUMMARY AND CONCLUSIONS

1. Myxedema heart is a manifestation of prolonged, severe hypothyroidism. It consists of enlargement of the cardiac shadow, diminished amplitude of contractions, and electrocardio-

graphic abnormalities with return to normal after thyroid treatment.

2. Myxedema heart is associated with pericardial effusions, cardiac dilation, and interstitial edema of the myocardium. These abnormalities may be due to the interstitial deposition of osmotically active colloid substances. Increased capillary permeability may be another factor of importance.

3. Hypercholesteremia and a high incidence of arteriosclerotic heart disease are present in myxedematous patients. The relationship of hypercholesteremia to atherosclerosis, however, has not been definitely established.

4. In a group of predominantly male patients with myxedema, the incidence of symptomatic arteriosclerotic heart disease was 44 per cent. Angina pectoris and myocardial infarctions were not uncommon in untreated myxedematous patients. Two patients had combined myxedema heart and coronary artery sclerosis. Myxedema heart without coronary sclerosis was not found.

5. Patients with and without symptomatic arteriosclerotic heart disease had similar cholesterol concentrations and thyroid function studies.

6. Thyroid treatment should be initiated at low levels and increased gradually. The presence of angina pectoris or myocardial infarction is not a contraindication to careful thyroid therapy.

The author wishes to express his appreciation to Drs. Reuben Bernan and Leslie Zieve, who reviewed this paper and offered many helpful suggestions.

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WHEN signs of congestive heart failure and cardiomegaly persist despite conventional therapy, long-term bed rest and hospital care specifically designed to lessen the work requirements of the heart may be successful. Control of heart rate and blood pressure by such treatment diminishes the burdens imposed by enlargement.

Patients selected for the program must demonstrate a willingness to cooperate. Strict bed rest is maintained, in addition to the usual therapy for chronic congestive failure. Initially, bathroom privileges are limited to a bedside commode. Patients showing improvement are permitted to walk only as far as a lavatory immediately adjoining the unit. The rooms are air conditioned, because a hot, humid environment increases cardiac work, cardiac output, and stroke volume. Adequate nursing services are essential. Mental and physical rest are attained only through social, occupational, and physical therapy in conjunction with complete nursing care.

Progress is usually slow the first few months, and gallop rhythm often persists. The patient also may be sensitive to digitalis preparations, and this will present difficulties in managing heart failure. However, after several months, improvement becomes more rapid, and the patient soon realizes the value of such therapy.

A group of 5 patients with extreme persistent cardiomegaly and low pulse pressure was treated and observed up to thirteen months. Extensive diagnostic studies failed to reveal the etiologic factors, and progressive deterioration on conventional therapy was obvious. Of the 5 patients, 2 were discharged from the hospital after ten and thirteen months of bed rest with normalsized hearts and without signs of heart disease. The remaining 3 patients have had steady improvement.

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Charles Edward Proshek, M.D. 1893-1957

WALTER H. JUDD, M.D.

Minneapolis

ONE OF THE MOST VERSATILE MEN the medical profession in Minnesota has known was Dr. Charles E. Proshek. He was a synthesis of general practitioner extraordinary, competent surgeon and obstetrician, gifted musician, world humanitarian, effective worker in practical politics, kindly gentleman, and cherished friend of all who were privileged to know him.

His combination of qualities was capped by such an unassuming humility that I am sure he never thought of himself as anything other than what every good physician and every appreciative American ought to be. It would be simpler, and equally accurate, to say only that he never thought of himself.

Charlie Proshek, as he was called by his friends, was born in New Prague, Minnesota, on September 4, 1893. He was 1 of 12 children, 6 of whom are still living. His parents had been brought as children from Czechoslovakia to the United States. The Proshek grandparents had established their homestead on a farm near New Prague around 1870.

As a youth, his interests centered principally on nature, sports, and music. The viola became his instrument, and, to the end of his life, he loved to spend a whole evening playing string quartets with friends.

As has been the case with so many other doctors, his desire to become a physician sprang from admiration of his home town physician, Dr. Edward Novak, one of the patriarchs of Minnesota's medical development. He was further drawn toward medicine by his sister, Antoinette, who had become a nurse.

Charles started his premedical studies at the University of Minnesota and was graduated from the medical school in 1917. After interning at the University and Minneapolis General hospitals, he moved to St. Cloud, Minnesota, where he practiced briefly until an appeal was issued by the American

Red Cross for volunteers for duty in war-devastated Eastern Europe. This he could not refuse. He was sent as a captain in the Red Cross to Siberia and there witnessed the struggle of the Russian people for true freedom and their betrayal by the communists. It was there, also, that he gave aid to the Czech Legionnaires as they fought their way across Russia and Siberia to Vladivostok.

Dr. Proshek's next two assignments were in Greece and Yugoslavia. Each of these governments honored him with high decorations — Knight of St. George, Greece, and Knight of St. Sava, Yugoslavia — for work done in the camps of World War I refugees. He later served in Poland in a similar capacity. It was this Red Cross relief work and the satisfaction he derived from it that set the pattern for his volunteering for similar work before and after World War II.

While in Europe, he met and in 1922 married Gabriela Preissova in Prague, Czechoslovakia. Mrs. Proshek came from a stimulating family. Her mother, Gabriela Preissova Halbaerthova, was a prominent and prolific writer and was considered one of Czechoslovakia's outstanding authors and patriots. Her novels were studied in the schools as classics and outstanding operas were based on several of them. Mrs. Proshek's two brothers, both lawyers, had government careers, one as a judge, the other as a government minister of state. With these highly cultured brothers-in-law, Dr. Proshek established a spontaneous and intimate friendship.

He prolonged his stay in Europe to study pathology at the University of Vienna. It was his plan to specialize in obstetrics and gynecology. On returning to the United States in 1922, he pursued this goal by working at the Chicago Lying-In Hospital and, later, in Minneapolis as an Adair fellow in obstetrics and gynecology. He was a member of the staff of the University of Minnesota and Swedish hospitals and, later, Minneapolis General Hospital.

The birth of his son, Lumir Charles, in 1923, and pressing financial needs forced him to continue general practice. Besides, he never was able or wanted to deny anyone who came to him for help, medical or otherwise—and they were myriad.

In 1928, Dr. Proshék accepted an appointment as Honorary Czechoslovak Consul for the Northwest: Minnesota, North and South Dakota, and Montana. In 1947, when the communists seized control of Czechoslovakia by treacherous coup, he indignantly resigned the post he had been so honored to accept from the bona fide government of Czechoslovakia nineteen years before. He kept up close contacts, however, even when necessarily clandestine, with key scholars, leaders, and patriots in Czechoslovakia.

Prior to World War II, he and Mrs. Proshék carried on almost a 2-person campaign to bring young Czech scholars to the United States. This was a desperate attempt to save at least a few choice people from the Nazi tyranny which they saw coming. They enabled 50 or more such students to further their education here with special attention to American methods, looking forward to the hoped-for time when they might be able to return to their homeland and assist in its rehabilitation and development as "the best democracy in Eastern Europe."

Charles did not lose sight of the "big picture," however, and, with the start of World War II, his attention turned more to our own national problems and policies. He recognized early that the United States could no longer live in security by itself alone. He saw what the disastrous effects on America would be if Europe were to be enslaved or crushed.

It was at this time that I came back to the United States from ten years of medical work in China with similar convictions, especially regarding the importance to this country of events developing in Asia. Our common convictions brought Dr. Proshék and me together in a kinship of spirit that grew with the years.

Whenever he developed an interest, it always resulted in action. In February 1942, he arranged, without my knowledge, a luncheon at which the first suggestion was made that I enter active political life to try to carry out our common convictions regarding our nation's peril.

World War II came to a close. The dreadful plight of the people displaced by the war and the hundreds of thousands more fleeing from their Communist-seized homelands became known to the world. Dr. and Mrs. Proshék established extensive correspondence with refugees from Czechoslovakia and lifted their morale with letters of hope and thousands of relief packages personally purchased, packed, and shipped. A new drive was launched by them to accelerate resettlement in the free world of the thou-

sands of superior persons languishing in the camps. Not a few were assisted in coming to the United States, where they have been a very worthwhile addition to the intelligentsia of this country.

In 1946, Dr. Proshék told me that he thought he was "good for one more big job." Was there need for a man like him to help in rescuing the hungry and dislocated peoples of Europe? There was, so he gave up his extensive practice and volunteered his services to the United Nations Relief and Rehabilitation Administration (UNRRA). He was sent to Germany where he helped supervise the establishment and management of some 80 camps for displaced persons.

This year and a half of duty, his last overseas, took a lot out of him. He saw afresh the age-old hatreds and prejudices of Europe, skillfully stirred up by the communists. Therefore, when he returned to medical practice in Minneapolis, at a time when many were predicting a great era of relaxation and peace, he knew better—and characteristically kept up his efforts, but never obtrusively, to alert his fellow citizens to the powerful, divisive forces at work in our world. He continued to work in local politics, searching for good men and women to support as candidates for public office. He gave generously of his own funds for such purposes and effectively persuaded others to do likewise.

He was active on several committees of the Hennepin County and Minnesota State Medical associations. One of the causes for which he worked was the establishment of a medical examiners' system in Minnesota. His work and research in this field had to be turned over later to others to be carried on.

In 1954, his practice was brought to an abrupt halt by a coronary thrombosis. However, before long he was able to resume it and his other interests to a limited degree, until he succumbed to a second thrombosis on the evening of October 30, 1957.

We are not apt to see again soon the like of Charles Proshék—so able but so modest, so determined and dauntless but so gentle, so devoted to his practice and patients and to a dozen other things at the same time, so intensely concerned about wrong and cruelty in the world but never himself hurting anyone, and doing so much for others while seeking so little for himself.

Here was a man whose active medical career, demanding as such a career always is, was only one of the many ways by which he worked for better people, better government, and a better world.

His son, Dr. Lumir Proshék, who has followed his father into medicine, recalls another doctor's saying to him some years ago, "If every doctor practiced medicine as your father did, this would indeed be a fine profession."

News Briefs . . .

North Dakota

DR. JOSEPH L. NOSAL has associated with Dr. Andre H. Lamal to form the MacKenzie County Clinic in Watford City. The new clinic will be located on the ground floor of the MacKenzie County Memorial Hospital.

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DR. TEOFI. EVANGELISTA has begun practice in the new medical center at McClusky. A native of the Philippines, he is a graduate of the St. Thomas University Medical School at Manila and has served at the Deaconness Evangelical Hospital in Milwaukee and the Bismarck Hospital. He will be assisted by his wife, who received nurse's training in Manila.

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DR. WILLIAM KITTO, formerly a pediatrician at the Northwest Clinic in Minot, has accepted a position in clinical investigation with the Parke Davis Company in Ann Arbor, Michigan. Dr. Kitto came to Minot in 1953 from Fort Collins, Colorado, and received his education at the University of Colorado at Boulder.

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DR. ROBERT BLATHERWICK has moved his practice to new quarters in Parshall, occupying a building constructed under his direction. His office was formerly situated above the Parshall Drug Store.

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DR. CECIL G. BAKER, formerly superintendent of the state hospital at Yankton, South Dakota, and a board certified psychiatrist, and Dr. James L. McDonald, a psychologist formerly of Hibbing, Minnesota, have formed the first psychiatric team in private practice to be established in North Dakota. They are located in the Bismarck Psychiatric Clinic, serving the Bismarck-Mandan area.

Minnesota

DR. HERMAN J. MOERSCH, senior consultant in medicine at the Mayo Clinic, retired from active practice in April and has accepted the position of director of education and research for the American College of Chest Physicians. Also retiring in April was Dr. Thomas B. Magath, a member of the clinic staff since 1921, who has taken up residence in Sarasota, Florida.

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DR. FRANCIS TYCE, a psychiatrist on the staff of the Rochester State Hospital, has accepted the position of acting clinical director of that institution, replacing Dr. Magnus C. Peterson, retiring director. Born in England, Dr. Tyce received his medical education there and was in private general practice until coming to America in July 1956 as a Fellow in psychiatry at the Mayo Foundation.

DR. D. W. DAVIES, a general practitioner, is the newest addition to the staff of the East Range Clinics at Eveleth. Following graduation from McGill University Medical School, Dr. Davies interned at St. Mary's Hospital in Montreal and served a one-year residency in pathology at Queen's University, Kingston, Ontario. His former practice was located in Harvey.

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DR. C. W. TRUESDALE of Glencoe has become the first member of the medical profession to be appointed to the Governor's Citizens' Committee on Aging, a group organized for the purpose of evaluating and reporting on nursing homes. Dr. Truesdale will serve on the nursing home classification subcommittee.

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DR. DUANE PANSEGRAU will join the staff of the Karlstad Hospital in the fall of this year. A native of Sioux City, Iowa, Dr. Pansegrau received his education at Iowa State University and is currently completing a tour of duty with the Air Force.

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DR. L. H. WITROCK has been named president of the Stearns-Benton Medical Society, serving all physicians residing in the two counties.

Deaths . . .

DR. R. K. DIXON, 61, died February 19 while en route from his home in Detroit to San Antonio, Texas. Formerly a member of the Mayo Clinic staff, Dr. Dixon was a native of St. Charles and a graduate of the University of Minnesota. After internship at Providence Hospital in Detroit, he served with the Mayo Clinic until 1932.

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DR. ESKIL ERICKSON, 59, died February 19 in Fargo as the result of a cerebral hemorrhage. A graduate of the University of Minnesota, Dr. Erickson served his internship at Ancker Hospital in St. Paul and was later a member of the staffs of St. John's Hospital in Fargo, St. Ansgar's Hospital in Moorhead, Ada Hospital, Hillsboro Community Hospital, and Bethesda and St. Francis hospitals in Crookston.

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DR. F. E. WHEELON, 85, died February 23 in Lake City, Florida. Long known as Minot's "family doctor," he was a graduate of the University of Minnesota and served as director of the First District Health Unit from 1946 to 1957. Dr. Wheelon was a member of the Northwest District and North Dakota medical associations.

The Journal Lancet

SERVING THE MEDICAL PROFESSION OF MINNESOTA,
NORTH DAKOTA, SOUTH DAKOTA, AND MONTANA

Series on NEUROLOGY for the PRACTITIONER

Cerebral Localization in the Practice of Medicine

A. B. BAKER, M.D.

Minneapolis

MOST PHYSICIANS deliberately avoid localizing lesions within the nervous system and, as a result, often feel insecure in their interpretation of neurologic illness. This no doubt is due to the fact that they continue to recall the complex intermixture of fibers and cells that plagued them during their undergraduate courses in neuroanatomy. It is certainly true that the nervous system is by far the most complex of all structures and does consist of a maze of cells and fibers with an inconceivable number of interconnections. However, from the point of view of practical application to clinical medicine, only a very few of these systems are used, and these offer the physician a relatively simple, easily applied approach to the problems presented by his patient. Since localization of lesions within the nervous system is often necessary to determine the nature and extent of a lesion and hence its therapy, it is of advantage to the physician to have some simple guides to such localization. Only those systems that are most frequently encountered will be discussed.

MOTOR SYSTEM

In nervous system diseases, motor system involvement is probably the most common. The

motor system consists of two parts, an upper motor, or pyramidal system, beginning in the motor cortex of the brain and a lower motor system beginning in the motor cells of the spinal cord.

Upper motor system

Clinical characteristics. Patients with involvement of the pyramidal system have chiefly an increase in muscle tone. This produces a slowing of muscle activity and results in a large number of complaints which may be misleading. These patients often complain of weakness, numbness, loss of feeling, awkwardness, scuffing the toe of shoes, or difficulty in descending stairs. Only by examination can the exact nature of the lesion be determined. The following were noted in an upper motor neurone lesion on examination:

1. Usually one-half of the body (hemiparesis) is involved when the brain is implicated and both limbs (diplegia, paraplegia) when the spinal cord is involved.

2. The deep reflexes—biceps, triceps, and knee jerks—are increased.

3. Muscle tone is increased as revealed by palpation or on moving the limb.

4. On plantar stimulation, the large toe on the involved side extends upward (Babinski's reflex).

5. In younger individuals, the abdominal reflexes are reduced.

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Clinicoanatomic relationships. This system begins from a wide area of the frontal and parietal cortex known as the motor cortex and then descends in an almost vertical fashion to and through the spinal cord (figure 1). At its onset, it is spread over a wide area of the cortex, but, as it descends toward the internal capsule, the entire system narrows to a small compact band of fibers and remains small throughout its course in the brain stem and spinal cord (figure 1). In the lower medulla, the tract crosses to the opposite side. In its course, this system lies adjacent to other functional areas, and the associated implication of these areas usually indicates where along the pyramidal system the lesion actually exists. Some of the more common of these landmarks may prove useful.

1. Motor cortex (1 in figure 1). Here the motor system is spread over a wide area. It is therefore difficult for a lesion to destroy the entire system without resulting in severe coma or death of the patient. Therefore, the usual lesion in the motor cortex results in involvement of a *single limb* rather than a hemiplegia or hemiparesis.

2. Frontal lobe lesions can extend posteriorly and implicate the motor system (2 in figure 1). These patients often show mild pyramidal involvement and, in addition, some *personality changes* due to frontal lobe damage.

3. Internal capsule or its vicinity (3 in figure 1). In this region, the motor system has narrowed to a small band. Its involvement usually destroys the entire system, resulting in a *complete hemiplegia* or *severe hemiparesis*. Since the sensory fibers are in close proximity in this region, the patient usually also shows a one-sided *sensory impairment*. If the lesion is in the left cerebral hemisphere, it will also be adjacent to the speech area and will be associated with an *expressive speech disturbance*.

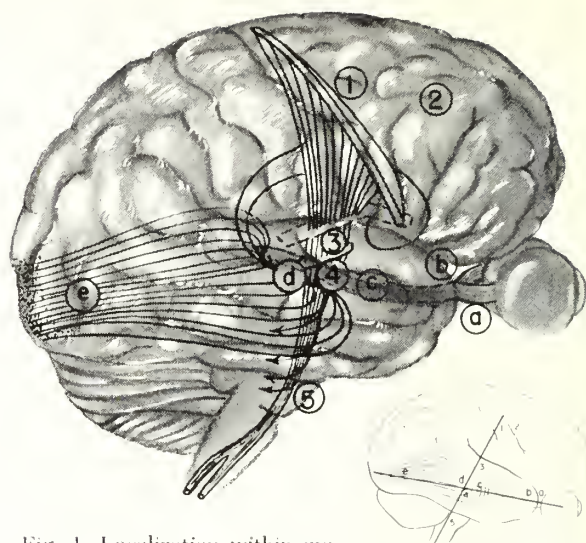


Fig. 1. Localization within motor and visual systems. *Motor system:* (1) cortex—monoplegia; (2) prefrontal area—weakness and personality change; (3) internal capsule—complete hemiplegia; (4) deep temporal lobe—hemianopsia and motor weakness; (5) brain stem—weakness and cranial nerve palsies. *Sensory system:* (a) optic nerve—one eye involvement; (b) optic chiasm—nasal or temporal anopsia; (c) beginning optic radiation—incongruous hemianopsia; (e) visual cortex—congruous hemianopsia.

4. Brain stem (5 in figure 1). The brain stem consists of the midbrain, pons, and medulla and contains the cranial nerve nuclei. Therefore, when the pyramidal system is involved in the brain stem, in addition to the *spastic hemiparesis*, there is also associated *cranial nerve involvement*. The level of the brain stem involvement can be determined by the cranial nerves that are implicated.

5. Spinal cord. This structure is so small that, when it becomes involved, the pyramidal systems on both sides become damaged, resulting in symptoms in both legs or all four limbs rather

TABLE 1
MOTOR INVOLVEMENT

Location of Lesion	Extent of motor involvement	Impairment of consciousness	Associated findings	Prognosis
Motor cortex	Monoparesis	Often present	Hemihypesthesia	Good
Internal capsule (deep)	Hemiplegia or hemiparesis	Usually none	Motor aphasia (if left sided)	Poor
Frontal lobe	Monoparesis	Often present	Mental and personality changes	Good
Brain stem	Hemiparesis	None	Cranial nerve involvement	Poor
Upper cervical cord	Quadripareisis	None		Poor
Lower cervical cord	Quadripareisis	None	Marked weakness of upper limbs	Poor
Thoracic cord	Diplegia or paraparesis	None		Poor
Lumbosacral cord	Monoparesis	None	Flaccid weakness of lower limb muscles	Poor

than involvement of one side of the body. Involvement of the thoracic cord results in diplegia, while implication of the cervical cord produces spasticity of all four limbs.

Lower motor system

This system begins in the anterior horn cells of the spinal cord and extends along the peripheral nerves to the muscles.

Clinical features. The most characteristic feature of this type of lesion is muscle weakness, which usually involves isolated muscles or muscle groups. This weakness is associated with a loss of muscle tone. The involved limb becomes flaccid. Atrophy of involved muscles occurs early, and the deep reflexes disappear or are reduced.

Clinicoanatomic relationships. Each of the muscles of the body receive innervation from specific motor cells in the spinal cord. By observing the location of the weakness and atrophy, the exact level of the spinal cord involvement can usually be determined. The following guides may prove useful:

1. Weakness of shoulder girdle indicates involvement of the fourth to the sixth cervical cord level.
2. Weakness of hands suggests involvement of the lower cervical cord level.
3. Weakness of pelvic girdle indicates involvement of the upper lumbar cord.
4. Weakness of foot muscles indicates involvement of the lower lumbar and upper sacral cord level.

A summary of motor involvement is presented in table 1.

VISUAL SYSTEM

The visual system, in contrast to the vertical orientation of the motor system, traverses the nervous system in a horizontal direction (figure 1). It, therefore, is an excellent system to use in cerebral localization, particularly in conjunction with the motor system.

Clinical features. Involvement of the visual system results in a loss of vision in certain segments of the visual field of one or both eyes. The nature of this field loss usually determines the location of the lesion along the visual system and can usually be determined by perimetric examination.

Clinicoanatomic relationships. The visual system begins in the retina of each eye and extends posteriorly to the optic nerves. Each optic nerve contains all of the visual fibers from the ipsilateral

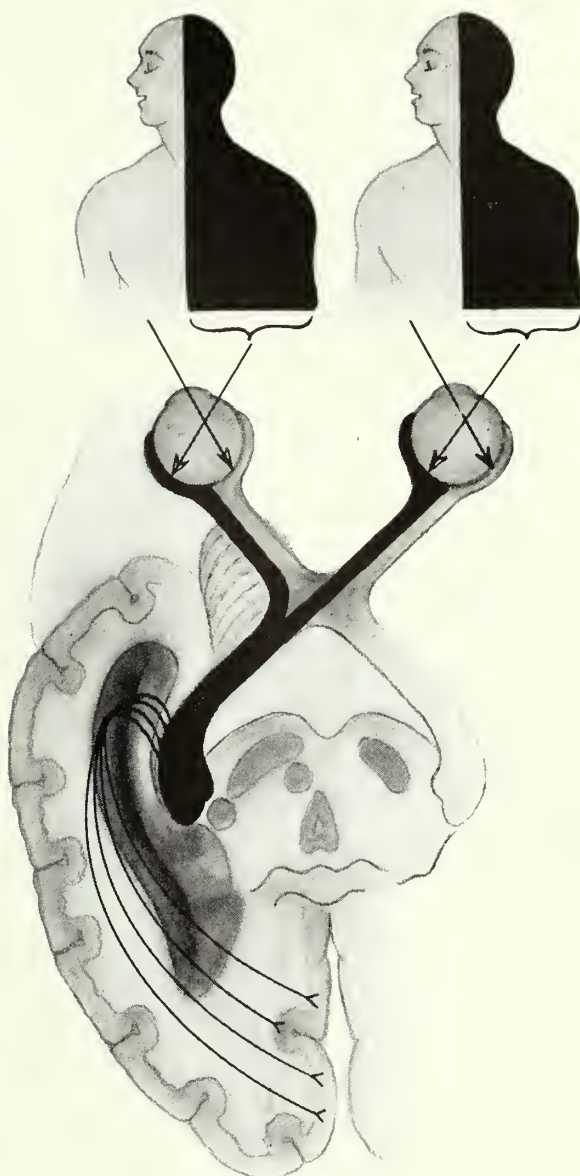


Fig. 2. Visual system illustrating crossing of fibers in the optic chiasm. Note contralateral visual field loss (homonymous hemianopsia) by a lesion of the visual system behind the chiasm.

al eye. Lesions of the optic nerve, therefore, produce visual disturbances in one eye only.

As the optic nerve passes into the optic chiasm, the visual fibers on the lateral half of the retina continue posteriorly on the same side to pass into the optic tract, the optic radiation and visual occipital cortex of the same side (figure 2). The medial part of the optic nerve fibers cross to the opposite side in the chiasm and pass posteriorly in the radiations to the opposite visual cortex (figure 2).

In view of this anatomy, the following visual

disturbances can be anticipated with lesions in different portions of the visual system:

1. Optic nerve (figure 1a). Lesions here produce impaired vision in one eye only, usually in the form of scotomata.

2. Optic chiasm (figure 1b). In this portion of the visual system, the fibers from the medial retina cross to the opposite side and comprise the medial portion of the chiasm. Lesions in this portion of the chiasm, such as pituitary tumors, will destroy these medial retinal fibers and result in a *bitemporal field loss* (figure 2)—loss of temporal or lateral field of vision in each eye. Lesions of the outer or lateral portion of the chiasm, such as sphenoid ridge meningiomas, will injure the fibers of the lateral retina, resulting in a nasal field loss in the involved eye (figure 2).

3. Optic tract and first part of the radiation (figure 1c). This part of the visual system contains fibers from the lateral retina of the ipsilateral eye, medial field of vision, and the medial retina of opposite eye, lateral field of vision. This results in an impairment of the half field of vision of each eye on the side opposite the lesion. This is referred to as a *homonymous hemianopsia* and is named according to the field of vision impaired rather than the involved retina (figure 2). Since the visual fibers in this part of the visual system are just beginning to organize after having crossed, the visual field loss is often unequal in the two eyes and is referred to as an *incongruous homonymous hemianopsia*.

4. Optic radiations (occipital, parietal lobe) (figure 1e). Lesions here also produce a homonymous hemianopsia. However, the visual fibers have now become well organized after crossing, so that the field loss in each eye is very uniform and is referred to as a *congruous homonymous hemianopsia*.

THE CRANIAL NERVES

Involvement of the cranial nerves (small figures in figure 3) offer one of the most accurate systems for cerebral localization. When present, it indicates specific localization to that part of the brain in which the cranial nerves or their nuclei are situated. Since the cranial nerves are distributed along the base of the brain, their involvement offers a means of localizing neurologic lesions in this large region of the nervous system. Only the most common cranial nerve disturbances will be discussed.

Olfactory nerve. This is the nerve of smell and is situated beneath the frontal lobes. Its involve-

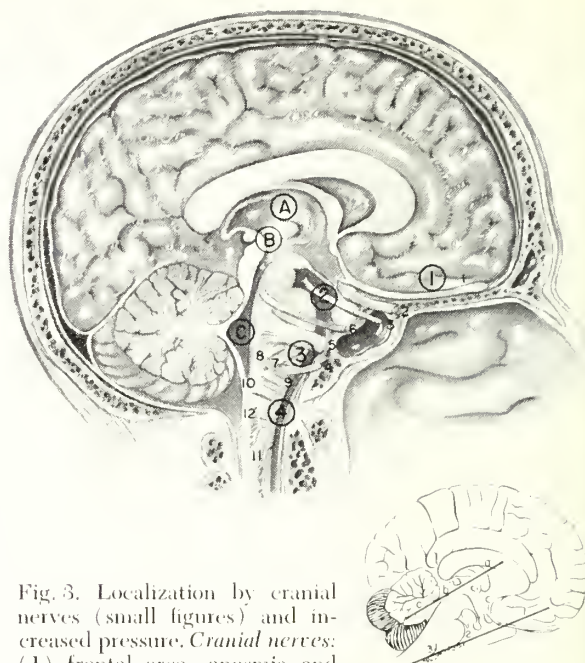


Fig. 3. Localization by cranial nerves (small figures) and increased pressure. **Cranial nerves:** (1) frontal area—anosmia and mental changes; (2) middle fossa—ocular palsies; (3) cerebellopontine angle—vertigo, hearing loss, tinnitus, and facial weakness; (4) brainstem—dysphagia. **Increased pressure:** (a) third ventricle—increased pressure only; (b) aqueduct region—increased pressure, impaired upward gaze, and ocular palsies; (c) fourth ventricle—increased pressure and incoordination.

ment produces an *anosmia*. Loss of smell alone is an unsatisfactory localizing finding because of the many local conditions within the nose that can interfere with smell.

Clinical syndromes. A unilateral loss of smell associated with mental changes should suggest a lesion of the inferior frontal lobe (1 in figure 3). This commonly is a meningioma, which, if diagnosed early, is readily amenable to therapy.

Oculomotor nerves, third, fourth, and sixth. These cranial nerves regulate eye movement and their involvement results in *double vision* or *ocular imbalance*. These cranial nerves have a long course along the base of the brain, passing from the brain stem to the orbit to supply the extraocular muscles. In their course, they pass along the tip of the temporal bone, the pituitary fossa, and the internal carotid artery, so that lesions of these structures can impair the function of these cranial nerves.

Clinical syndromes. The sudden onset of diplopia or ocular imbalance should suggest some vascular process, such as an aneurysm, particularly of the internal carotid artery. Occasionally, extension of a carcinoma from the sinuses produces acute third nerve palsy.

A more gradual involvement of these cranial nerves can result from a neoplastic process, such as a meningioma of the temporal bone or the sphenoid ridge (2 in figure 3).

Trigeminal nerve. The fifth cranial nerve is a sensory nerve to the face and its involvement produces either facial pain or facial hypesthesia. The nerve originates in the pons, and its ganglion is situated along the base of the brain in the middle cranial fossa. Involvement of the ganglia results in facial pain. Involvement of the pontine nucleus usually results in impaired facial sensation.

Clinical syndromes. Unilateral facial pain, particularly if accompanied by ocular palsies, indicates a lesion along the base of the brain in the region of the middle cranial fossa. Such a lesion often consists of an aneurysm of the internal carotid artery or a metastatic carcinoma from the underlying sinuses.

Facial hypesthesia suggests a lesion involving the pontine region more posteriorly and is usually a tumor within or outside the brain stem.

Facial nerve. This is a motor nerve of the face, and its involvement results in a weakness of all the facial muscles on one side. Its nucleus is situated in the upper medulla and leaves the brain stem in close proximity to the eighth cranial nerve (auditory, vestibular). After leaving the brain stem, the facial nerve takes a long course through the bony facial canal to reach the muscles of the face.

Clinical syndromes. Isolated weakness of the facial muscles on one side usually suggests an involvement of the facial nerve in the facial canal and is known as Bell's palsy.

Along the brain stem, the facial nerve is implicated with the eighth cranial nerve, resulting not only in facial weakness but also in vertigo, tinnitus, and hearing loss. Such findings suggest a lesion at the upper part of the medulla in the cerebellopontine angle (3 in figure 3).

Lower cranial nerves, ninth, tenth, and twelfth. These cranial nerves extend from the lower medulla to the musculature of the pharynx, larynx, and tongue. Their involvement is manifested clinically by difficulty in swallowing and talking.

Clinical syndromes. Any patient with difficulty in swallowing and talking has a lesion of the lower medulla (4 in figure 3). This syndrome has been labeled bulbar palsy. The difficulty in swallowing may result in obstruction of the airway by secretions and may jeopardize the life of the patient. Hence, acute onset of such a syndrome often comprises a medical emergency.

In the younger age groups, the acute onset of bulbar palsy should suggest poliomyelitis, a Guillain-Barré syndrome, or myasthenia gravis. A more chronic lesion usually indicates a brain stem tumor.

In older individuals, bulbar palsy is most commonly due to vascular or degenerative processes, such as amyotrophic lateral sclerosis, pseudobulbar palsy, or a basilar artery insufficiency.

A summary of cranial nerve involvement is presented in table 2.

ACUTE INCREASED INTRACRANIAL PRESSURE

The symptoms and signs of increased intracranial pressure are chiefly headache, nausea, and papilledema. When these findings occur acutely,

TABLE 2
CRANIAL NERVE INVOLVEMENT

Nerve	Clinical symptoms	Clinical syndrome	Type of lesion
Olfactory	Anosmia	Anosmia and mental symptomatology	Frontal lobe meningioma
Oculomotor	Diplopia or ocular palsy	Acute onset	Aneurysm of internal carotid artery or carcinoma of sinus
		Chronic onset	Meningioma of sphenoid ridge or temporal bone
Trigeminal	Facial pain	Facial pain with ocular palsy	Aneurysm of internal carotid artery
	Hypesthesia	Facial hypesthesia	Pontine tumor
Facial	Facial weakness	Facial weakness only	Bell's palsy
		Facial weakness, tinnitus, and vertigo	Cerebellopontine angle tumor
Lower cranial nerves	Dysphagia and dysarthria	Acute	Polio, Guillain-Barré syndrome, myasthenia gravis, or vascular processes
		Chronic	Tumors or amyotrophic lateral sclerosis

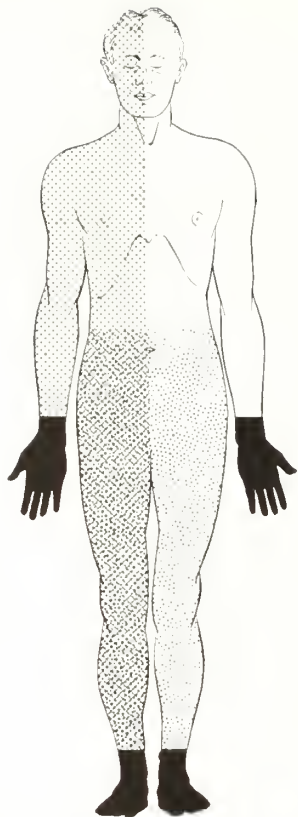


Fig. 4. Sensory disturbances. Hatched lines reveal the typical hemihypesthesia as seen in thalamic lesions. Dotted area shows segmental sensory involvement of spinal cord lesions. Solid black areas demonstrate the "glove-stocking" involvement of peripheral nerve lesions.

they generally indicate an obstruction to the flow of cerebrospinal fluid. Since the flow of cerebrospinal fluid is generally blocked in that part of the ventricular system which is situated deep along the midline of the brain, the acute onset of such increased pressure almost always suggests the presence of a deep-seated midline lesion. This symptoms-complex can, therefore, be used to localize lesions along the deeper areas within the brain.

Third ventricle. This region is relatively large, and lesions here may become fairly extensive without implicating other structures. When other structures are involved, they are usually the basal nuclei which are situated lateral to the third ventricle.

Clinical syndromes. A lesion within the third ventricle results only in signs of acute increased intracranial pressure (figure 3a). Additional evidence of basal ganglia involvement, such as tremor or dyskinetic movements, is rare.

Midbrain. After leaving the third ventricle, the spinal fluid passes through the aqueduct

within the midbrain. In this region are situated the nuclei of the oculomotor nerves. A lesion in this area would not only obstruct the flow of spinal fluid but would also implicate the oculomotor system (figure 3b).

Clinical syndromes. Midbrain lesions result in the symptoms and signs of acute increased intracranial pressure accompanied by diplopia, ptosis, ocular palsies, and an inability to look upward. The most common lesion in this region producing this combination of symptoms is the pinealoma, which occurs in younger individuals.

Posterior fossa (cerebellum). In this region, the spinal fluid flow is through the cerebellum. Any lesion causing obstruction to the fluid flow usually also results in cerebellar symptomatology (figure 3c).

Clinical syndromes. Lesions involving the cerebellar hemispheres result in incoordination of the upper limbs and ataxia. In midline lesions, the patients show trunkal ataxia. The cerebellar lesions resulting in increased pressure are invariably tumors and are seen mostly in children.

The remaining neurologic systems are, as a rule, too inconsistent to be used in cerebral localization. Seizure manifestations can often help greatly in localizing lesions within the brain, but these localizing features will be described in a later paper on the convulsive disorders. Brief comment might be made on the significance of sensory disturbances in cerebral localization. Although not too accurate in localizing lesions to specific areas of the nervous system, sensory impairment may be helpful in determining the gross area involved.

Sensory cortex. A lesion in this area produces a unilateral impairment of vibration or of position sense with associated astereognosis and a loss of two point discrimination. The involvement implicates one half of the body and often spares the face.

Thalamus. Involvement in this region characteristically produces a complete hypesthesia involving one half of the body (figure 4). Often, this sensory disturbance is mild and not offered as a complaint by the patient.

Spinal cord. A lesion of the spinal cord produces a segmental sensory loss. There is usually a loss of sensation below the involved level, and this sensory loss implicates both sides of the body (figure 4).

Peripheral nerve. A glove-stocking type of sensory impairment characterizes peripheral nerve lesions. The sensory disturbance implicates the fingers or toes and extends proximally in a band-like fashion (figure 4).

Recent Developments in Ophthalmology

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AS IN OTHER FIELDS of medical science, the field of ophthalmology is literally bursting with new ideas. Each month an ever-increasing stream of articles flows from the pens of ophthalmologists and allied research workers all over the world. It is the purpose of this paper to present a brief synopsis of some of the newer developments in ophthalmology so that others outside this field may gain more insight into modern ophthalmologic trends.

Of necessity, this synopsis must be short and therefore incomplete. Major developments will not be omitted, however, though many developments may be cited with no discussion. I would also like to make it clear that many of the things listed do not necessarily represent permanent advances in the field of ophthalmology. Many have had very limited or no clinical application, may not stand the test of time, and do not at present belong in the practicing ophthalmologists' armamentarium. A few, which I shall try to note, promise to revolutionize or have already revolutionized certain phases of ophthalmology.

I shall divide the subject into New Drugs and New Instruments and Technics.

NEW DRUGS

Alphachymotrypsin. Probably the most dramatic discovery in the field of ophthalmology in recent years, this substance is at present the subject of conversation among all ophthalmologists. A proteolytic enzyme first introduced by Barraquer, a Spanish ophthalmologist, alphachymotrypsin "dissolves" the zonules holding the lens in place, thereby rendering its removal during cataract extraction much easier. Anyone who has used this compound knows that it works. Since younger people have stronger zonules than older individuals, it is of considerable help in the

group of cataract patients between 35 and 60 years of age. It makes intracapsular cataract extraction easier at all ages but, like all substances, it may have some drawbacks, particularly with respect to its effect upon the cornea. Extensive data are accumulating on the use of the drug, however, and its drawbacks will be more clearly delineated in the future. At present, its advantages appear to outweigh its disadvantages in many cases.

Carbonic anhydrase inhibitors. These drugs reduce aqueous humor secretion and thereby lower the intraocular pressure. They are administered by mouth and represent the first major breakthrough in control of the intraocular pressure since miotics. Acetazolamide (Diamox) and dichlorophenamide (Daranide) are two of the best-known members of this group. They are not successful in all glaucomas, and, unfortunately, tolerance often develops on prolonged administration. They are thus of most value for short-term reduction of the intraocular pressure.

New miotics. Demecarium bromide (Humer-sol) and echothiophate iodide (Phospholine iodide) are two new miotics so powerful that one drop of either one in a normal eye will produce miosis for months. Whether or not they will prove to be of much additional benefit in the control of the glaucomas is yet to be determined. Some previous strong miotics have been found to result in ciliary body edema, with resultant narrowing of the anterior chamber angle and an actual sudden rise in the intraocular pressure.

P32. Because radioactive phosphorus concentrates in intraocular tumors after systemic administration, this substance has proved useful as an adjunct in the diagnosis of these lesions. Use of a Geiger counter at the proper interval will reveal an increased count over the area of the tumor. However, this laboratory test must not replace shrewd clinical judgment in the diagnosis of intraocular neoplasms, as, in most cases,

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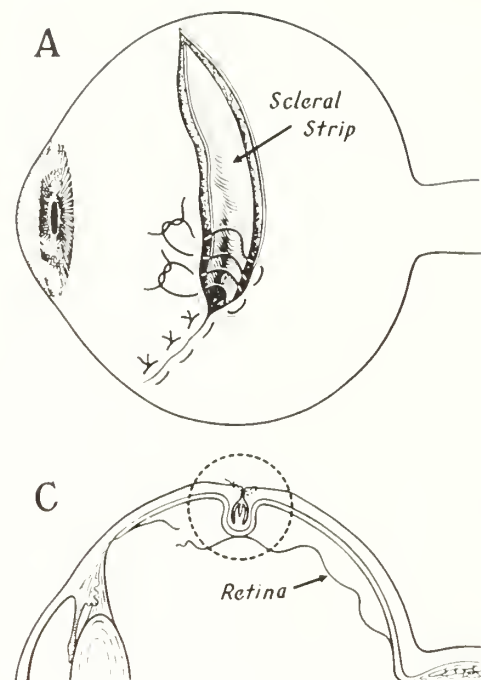


Fig. 1. Steps in the scleral buckling procedure utilizing a strip of buried sclera. Note that the ridge must be behind the retinal tear as illustrated in C.

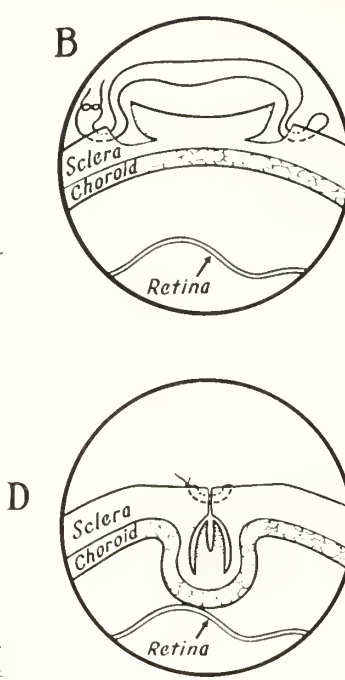


Fig. 2. Scleral buckling procedure utilizing a piece of buried polyethylene tubing

the most reliable means of diagnosis is still clinical examination.

NEW INSTRUMENTS AND TECHNIQUES

Scleral shortening and buckling operations for retinal detachments. Although performed on a limited scale many years ago, the widespread use of these operations is very recent and hence they merit discussion. Although there are many modifications of techniques, the basic principle of all of these operations is the same. The aim of all is either to shorten the globe, thereby reducing the area to which the detached retina must adhere, or to create an inward buckling of the sclera, thereby forming a ridge or dike inside the eye to which the detached retina can adhere.

The principles of two buckling procedures are illustrated in the accompanying illustrations. Figure 1 shows a scleral buckling procedure utilizing a strip of buried sclera. Note the ridge formed inside the eye. This ridge or dike must be behind the retinal tear to successfully seal the leak of vitreous through the retinal tear. Note the retinal tear anterior to the ridge in Figure 1c. Figure 2 shows a similar procedure except that in this case the scleral strip is excised and a

piece of polyethylene tube is buried permanently in its place.

In both of these procedures, diathermy is also used and drainage of subretinal fluid must be obtained. In fact, a large amount of subretinal fluid must be evacuated, as both procedures result in a considerable reduction in vitreous volume.

These procedures are not indicated in all retinal detachments by any means and, although in some cases they undoubtedly result in reattachments where conventional diathermy would or has failed, retinal detachments must still be looked upon as a most serious calamity with no magic therapeutic formula at hand.

Light coagulation of the retina and choroid. Two years ago, Professor Meyer-Schwickerath of Bonn, Germany, announced a remarkable machine by which he could focus a beam of light on the retina of such intensity that coagulation of the retina and underlying choroid would ensue. This destruction of a localized area in the fundus can be kept under direct observation by the operator during the entire procedure, giving pinpoint accuracy. The machine appears to have its greatest usefulness in the treatment of con-

ditions as Eales's disease and angiomas (von Hippel's disease) of the retina. It may be useful in the sealing of tears in the retina when the edges of the tear are in close apposition to the pigment epithelium of the retina. It will undoubtedly be tried in intraocular tumors, but whether or not it will succeed in this sphere has yet to be determined. The machine is very expensive, costing over \$12,000. This will delay its widespread acquisition.

The Berman locator. Although this device is not new, being first used at Pearl Harbor in the field of general surgery, recent improvements make its use now mandatory in removal of magnetic intraocular foreign bodies. Based upon the principle of a mine detector, the probe of this instrument detects buried magnetic foreign bodies with uncanny accuracy. Most intraocular foreign bodies are magnetic, and, therefore, its use in eye surgery for removal of these foreign bodies tells us exactly where to make the incision in the eye for removal of the foreign body with the least amount of tissue trauma. Its accuracy of localization exceeds that of x-ray and, furthermore, provides an up-to-the-minute localization during surgery, should the foreign body shift position. An audible signal tells the operator how near he is to the foreign body and he thus need not raise his eyes from the operative field. This instrument has truly revolutionized magnetic intraocular foreign body removal. It is also useful for detecting buried hypo needles elsewhere in the body.

Surgical needles. It may seem strange to include mere surgical needles as a new development in ophthalmology, but their inclusion is fully justified. The development of special cutting needles strictly for eye work has made cataract and other intraocular surgery safer. More than any other single factor, these new needles are responsible for early ambulation and the absence of sandbags on the side of the cataract patient's head. These new micropoint needles have an edge on them as sharp as a well-honed knife and may be passed through tough cornea or sclera with an ease not dreamed of a few years ago. Some of them are being made only 4 or 5 mm. long for delicate corneal work.

Keratome (microtome). A new mechanical device for shaving thin sections off the cornea has been developed. It resembles an electric shaver in appearance and sound and is of considerable help in corneal grafting where only a partial thickness of the cornea is to be grafted.

Microlenses. These small, wafer-thin contact lenses have almost completely replaced the old scleral contact lenses. They cause less corneal

clouding and are much more comfortable to wear than the older contact lenses.

Anterior chamber lenses. The previous intraocular lens of Ridley, which was inserted into the eye behind the iris after cataract extraction, has been largely abandoned. In its place, researchers are working on thin, plastic lenses which can be inserted into the anterior chamber. These lenses are held in place by short prongs which impinge on the angle of the anterior chamber. These lenses obviate the necessity of wearing the ordinarily thick cataract lenses that aphakic patients formerly had to wear. Their use as yet must be regarded as strictly experimental. While attempts to fit lenses inside the eye have been proceeding, conventional forward cataract lenses have been gradually improved, the latest improvement being a plastic, aspheric cataract lens which is thinner than the conventional cataract lens and provides better edge correction. An aspheric lens is one which has a continuously changing curve from the center to the periphery. These lenses have been the goal of optical experts for over two hundred years but only recently has mass production of them been accomplished.

Electromyography and electroretinography. It is now possible to record action potentials from individual ocular muscles and from the retina. Although both of these developments are primarily research tools at the moment, each technic promises the possibility of wider clinical application in the future. The ocular electromyogram is said to represent the most sensitive test for myasthenia gravis currently available.

Miscellaneous instruments and techniques. Space does not permit even a brief discussion of many new developments in ophthalmology, but the following should be listed: the Harrington screener for rapid visual field testing; the applanation tonometer for measurement of the intraocular pressure without applying appreciable pressure to the globe as conventional tonometers do; freeze-drying and preservation of ocular tissues, such as vitreous, cornea, and sclera; and ultrasonics for the detection of intraocular tumors.

SUMMARY

A very brief glimpse of some of the developments of modern ophthalmology has been provided. After causing a brief flurry of excitement, some may quickly succumb to the test of time. Others have already become firm cornerstones in present day ophthalmologic practice. Taken as a whole, these developments represent thrilling new vistas in the field of ophthalmology.

Rabies in North Dakota

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Grand Forks

FOR MANY YEARS, North Dakota had thought of itself as being free of rabies. This concept changed dramatically with the occurrence of a case of rabies in a skunk in the summer of 1950 near the town of Lisbon.

Actually, North Dakota has had periodic outbreaks of rabies in domestic as well as wild animals since its earliest settling. Well-authenticated outbreaks in skunks are mentioned in many of the historic novels of the early Great Plains area. Maria Sandoz, in her book, *Buffalo Hunters*,¹ refers to attacks on buffalo hunters by "hydrophobic" skunks. One of the earliest scientific references to rabies in North Dakota is made in the annual report of the Public Health Laboratories for the year 1911.

That report states, "During the spring of 1911, a considerable number of mad dogs were shot without having attacked any persons but a number of persons were bitten, four of which presented themselves for treatment in the Public Health Laboratories during the months of May and June."² In each instance, Negri bodies were demonstrated in the brain of the offending animals. Other references are made to rabies at periodic intervals during the public health history of North Dakota.

The ten years immediately preceding our present outbreak, which began in 1950, were free of any known cases of rabies.

In 1945, Iowa reported an alarming increase in the number of rabid skunks in southern Iowa. With each succeeding year, reports of positive animals were from areas progressively farther north. By 1950, an appreciable increase in the number of cases in Minnesota was being experienced. In May 1951, D. S. Fleming,³ chief, Section of Preventable Diseases, Minnesota State Health Department, had this to say: "We consider the rabies problem a very serious one at the present time and a very difficult one to handle. The disease appears to be widely spread

throughout the skunk population in the southern half of the state, and because it is now unrewarding economically to trap or destroy skunks, the disease will apparently continue to spread."

G. J. Van Hensvelen,⁴ state health officer of South Dakota, reported 9 animals positive for rabies in 1950 and 18 in 1951. In 1950, only 1 of the rabid animals was a skunk, while, in 1951, 12 were skunks. He further stated, "All the positive animals in the last two years have been in the eastern third of the state. Previous to 1950, the number of positive animals was insignificant."

This information left us with the implication that if skunk rabies was to continue to spread in a northwesterly direction, as it had been, North Dakota could soon expect to experience some type of a rabies outbreak in its large skunk population. This period of expectation reached its culmination with the first reported case of skunk rabies in the summer of 1950.

TABLE 1
ANIMALS EXAMINED FOR RABIES
FROM APRIL 7, 1951, TO DECEMBER 15, 1951

Animal	Positive	Negative	Suspicious	No diagnosis	Animal inoculation positive
Mouse	17	42	4	1	
Skunk	33	28	6		
Mink	4	4			
Muskrat	1	8		1	
Squirrel	2	5	1		
Rat	6	7	1	4	
Woodchuck	1				
Bobcat		1			
Fox	1	1			
Badger	1	2			
Raccoon	3	1	1		
Gopher	2	3	2		
Rabbit		1			
Dog	14	19	1		
Pig		2			
Bovine	5	3	1		
Cat	28	62	10		2
Sheep			2		
Total	118	189	29	6	2

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The spread of rabies into North Dakota can be traced in a fairly chronologic fashion, starting in 1945 in Iowa and gradually spreading into North Dakota by 1950 (figure 1). The spread has continued until almost all of the geographic areas of the state are now involved.

The year 1951 saw the greatest incidence of rabies that has ever been reported in a given year in North Dakota—118 cases (table 1). The laboratory diagnosis of rabies during 1951 left a great deal to be desired, as is evidenced by the 17 field mice that were reported as positive for rabies. In no instance has any case of rabies in field mice in North Dakota been satisfactorily confirmed by laboratory tests. In all probability, none of the 17 cases were actually rabies. During this period of alarm and anxiety, there appeared to be a tendency to overdiagnose; some of the laboratories involved were actually reporting cases as positive which were in reality negative.

This apparent inconsistency resulted in a request to the United States Public Health Service that a competent rabies expert be sent to North Dakota to study the situation. As a result of his study, it was reaffirmed that a rabies problem had developed and that some system of stand-

RABID ANIMALS North Dakota 1952 - '58

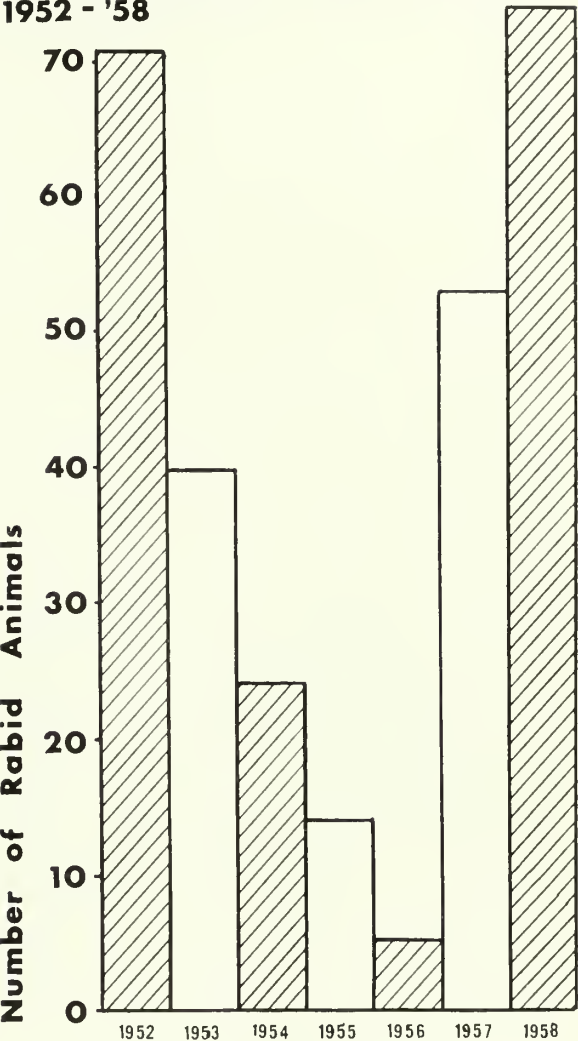


Figure 2

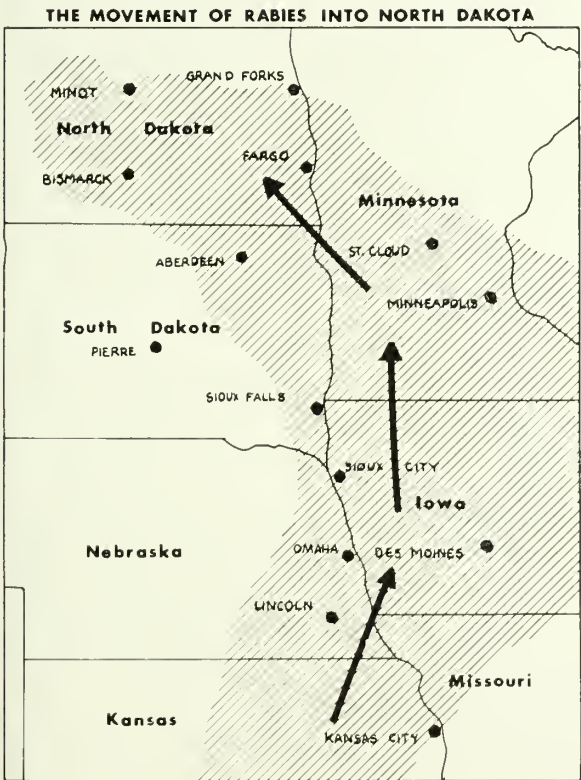


Figure 1

ardizing the laboratories involved in rabies diagnosis be instituted.

It was his recommendation that all cases of suspected rabies where there is a human exposure which are submitted to any laboratory in North Dakota be referred to the Grand Forks Public Health Laboratory for microscopic confirmation as well as for mouse inoculation procedures. This procedure was instituted on February 1, 1952. It resulted in a much lowered incidence of reported rabies during the years that followed.

RESULTS

In the seven years that followed, 1,088 animal heads were sent to the Public Health Labora-

TABLE 2
RABIES IN ANIMALS NORTH DAKOTA, 1952-1958

<i>Animal</i>	<i>1952</i>	<i>1953</i>	<i>1954</i>	<i>1955</i>	<i>1956</i>	<i>1957</i>	<i>1958</i>	<i>Total</i>
Skunk	23	19	8	8	5	40	47	150
Cattle	9	15	12	3	0	6	11	56
Cat	16	1	1	0	0	3	6	27
Dog	10	2	3	2	0	1	5	23
Fox	1	1	0	1	0	0	2	5
Swine	1	1	0	0	0	2	1	5
Sheep	1	0	0	0	0	0	0	1
Raccoon	1	0	0	0	0	0	0	1
Squirrel	1	1	0	0	0	0	0	2
Muskrat	1	0	0	0	0	0	1	2
Weasel	0	0	0	0	0	1	0	1
Gopher	0	0	0	0	0	0	1	1
Mink	2	0	0	0	0	0	0	2
Badger	2	0	0	0	0	0	0	2
Beaver	1	0	0	0	0	0	0	1
Rabbit	1	0	0	0	0	0	0	1
Deer	1	0	0	0	0	0	0	1
Total	71	40	24	14	5	53	74	281

tory, either directly or for confirmation by some other laboratory. From 1952 through 1959, 281 animal heads were reported positive for rabies by all laboratories; these were both human exposure and nonexposure animals. Most of these animals were skunks; followed by cattle, cats, and dogs, respectively. Thirteen other species of animals were involved, but only 1 or 2 of each species (see table 2). In this seven-year period, the peak incidence of rabies in North Dakota appeared to be during the years of 1952 and 1958 (see figure 2).

During the period of February 8, 1952, through March 15, 1959, a total of 45 rabies-positive animals in which there was human exposure were diagnosed either by microscopic smear or mouse inoculation test in the Public Health Laboratory. Thirty-eight of these were submitted from North Dakota, while 6 were sent in from Minnesota.

Of these 38 human exposures, 13 were cats, 12 skunks, 11 cattle, 2 dogs, and 1 sheep (table 3).

Of the 45 positive human exposure specimens done in the Public Health Laboratory, 27 were positive by microscopic examination and 1 was highly suspicious. Seventeen of these specimens were positive animals only by mouse inoculation test. In other words, 35.5 per cent of all positive animals were negative by microscopic examination. Of the 17 animals negative microscopically and positive by mouse inoculation test, 9 were cattle, 7 were cats, and 1 was a dog. The most frequent microscopic-negative, mouse inoculation-positive animals were cattle.

The incubation period for rabies of bovine origin in mice tended to be rather long, not less than fifteen days and as long as twenty-three days, with an average of eighteen days. At this point, the test animals died rapidly, usually

TABLE 3
HUMAN EXPOSURE SPECIMENS POSITIVE FOR RABIES
NORTH DAKOTA FEBRUARY 8, 1952, THROUGH MARCH 15, 1959

<i>Animal</i>	<i>1952</i>	<i>1953</i>	<i>1954</i>	<i>1955</i>	<i>1956</i>	<i>1957</i>	<i>1958</i>	<i>1959</i>	<i>Total</i>
Cat	6	0	0	0	0	2	5	0	13
Skunk	3	3	0	0	1	3	2	0	12
Cattle	0	3	0	0	0	2	3	3	11
Dogs	0	0	1	0	0	0	1	0	2
Sheep	1	0	0	0	0	0	0	0	1
Total	10	6	1	0	1	7	11	3	39

LOCATION OF HUMAN RABIES EXPOSURES **February 8, 1952 to March 15, 1959**

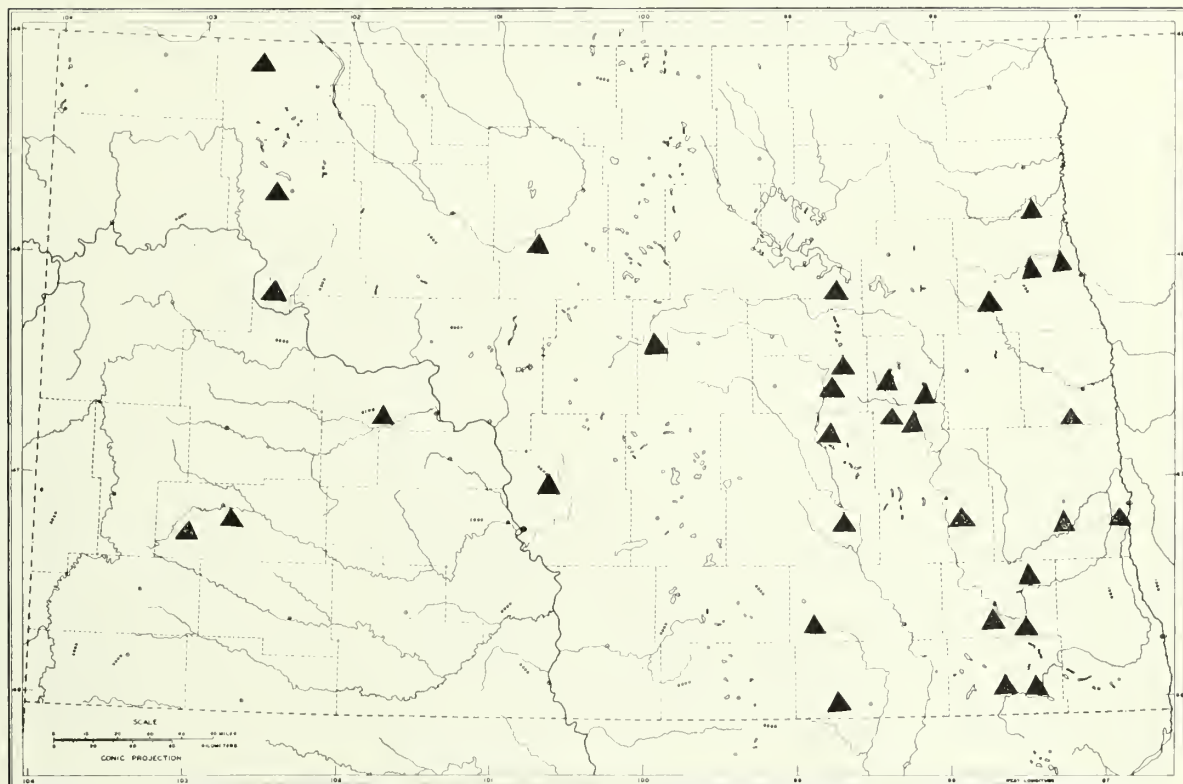


Figure 3

twenty-four to thirty-six hours after onset. Typical Negri bodies could be readily demonstrated in large numbers at this time. Reexamination of the original smears proved to be negative.

Human rabies exposures have occurred in 21 of North Dakota's 53 counties. All geographic and ecologic areas of North Dakota have been involved. Rabid animals have been found in almost all counties of North Dakota, but not all of them contributed to a human exposure. The greatest number of human exposures have occurred in the east central and southeastern portions of North Dakota. This may be accounted for in part by the fact that roughly two-thirds of the people of North Dakota live in the eastern one-third of the state. It is also interesting to note the relatively close proximity to the James and Sheyenne River systems in which eastern human exposures are found (figure 3). This is probably due to the heavily wooded river banks and coulees adjacent to the river, which offer good natural hiding places in a prairie region.

CONCLUSIONS

1. The largest recorded rabies outbreak in North Dakota occurred during the period of 1950 through 1959.

2. The introduction of rabies in this state was the result of a normal extension of an outbreak of skunk rabies which occurred in Iowa in 1945.

3. The most frequently involved animal and probably the most frequent carrier of rabies in North Dakota is the skunk.

4. Wild domestic cats have been implicated in more human exposures than any other species, although cattle and skunks are involved about as often.

5. Rabies appears to be a problem primarily of skunks, cats, and cattle. To date, only a very limited number of dogs have been involved.

6. Adequate rabies diagnosis must include animal inoculation tests in order to be complete.

7. There tends to be more human exposures in areas along river systems, probably because of the greater concentration of wild animals, especially skunks, in these areas of natural cover.

8. Rabies in North Dakota is largely a problem of wild, semiwild, or large domestic animals and not one of household pets.

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Nonpenetrating Chest Trauma

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THORACIC INJURIES are estimated to cause 25 per cent of immediate and delayed fatalities of automobile accidents. These injuries are either of a direct or a deceleration type of trauma.

Direct trauma results from the impact of the automobile frame and parts on the individual. In the violence of the crash, passengers may ricochet against each other or the many unyielding decorations of the automobile's interior or be dislodged at relatively high speed from the vehicle. Pedestrians involved in accidents are injured by the juggernaut force of the automobile.

Deceleration injuries are equally as serious. The various viscera react to the tremendous interplay of static and kinetic forces, the effect on each viscus being directly related to its mass, structure, location, and attachments. Certain areas of each organ are most subject to stress forces and liable to rupture. Contrecoup injuries also occur, as the thoracic viscera are similar to the brain within the calvarium. Pedestrians suffer similar injuries, usually from projectile accelerations, when struck.

External signs of serious intrathoracic damage may be misleadingly meager, especially if there are other more dramatic nonthoracic injuries. It is most important to realize that seemingly minor injuries to the chest may originate a serious disturbance of physiology with grave hazard to the patient's life. To facilitate descriptions, this discussion considers injuries under the various organs involved.

CHEST WALL

Fractures of the sternum, clavicle, scapula, or ribs are the most common injuries. Such isolated fractures plus soft tissue damage are painful, incapacitating, and of aggravating duration.

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If unassociated with other thoracic trauma, the most serious potential of these injuries is the detrimental effect on cardiorespiratory physiology because of pain. This pain precipitates improper cleansing of tracheobronchial secretions. "Wet lung" syndrome, atelectasis, and pneumonia tend to result, especially in the aged. If multiple segmental rib fractures, or flail chest, occur, these pulmonary complications are usually present in varying degrees.

Tracheobronchial secretions must be evacuated either by the patient's coughing or by artificial means, such as tracheotomy, bronchoscopy, or tracheal suction. Control of pain by analgesics, procaine block, or both, is all-important. Rib belts are still used, especially in the semiacute second and third weeks after injury. A flail chest must be stabilized externally, by skeletal traction; directly, by wiring the fractured ribs; or internally, by tracheostomy and Moreh respirator.¹⁻⁴

LUNG AND PLEURA

Pulmonary injury results from direct violence or contrecoup trauma. Direct violence may be seen as subpleural hemorrhagic imprints of ribs, pulmonary hematoma, or traumatic lung cavitation. Chronic hematoma is often discrete and confused with a neoplastic coin lesion. Although a history is exceedingly helpful, exploratory thoracotomy is indicated if the lesion does not regress in six to eight weeks. The traumatic disruption of the lung parenchyma with resultant peripheral elastic pull visualizes as a cavity surrounded by a small zone of atelectasis. Unless complicated by secondary infection, spontaneous healing is the rule.

Compression of the chest is usually in an anteroposterior plane, with rigid spine and mobile sternum. As a result, contrecoup lesions are usually posterior. The earliest stage is a straight line of pulmonary subpleural hemorrhage from apex to base opposite the angle of the ribs. Often, the cupula will be involved. This contusion may be bilateral, massive, and lethal. Multiple intrapulmonary hemorrhagic spots may be present.

Another type of lung injury is the "pincer" in the phrenicocostal areas. In these locations, the lungs are forcibly compressed in the V-shaped recesses with resultant alveolar hemorrhage. If the injury is extensive, fatal pulmonary edema may result.

The "wet lung" syndrome is essentially a sign and symptom of retained bronchial secretions; overproduction of pulmonary fluid from injury, that is, edema; secondary reaction of the pulmonary tissues to hemorrhagic diathesis; and inability of the individual to clear these cumulative fluids. A vicious cycle of anxiety, atelectasis, anoxia, and more fluid transudation develops. This syndrome may evolve from minor or major chest injuries. Improving expectoration by alleviating pain through use of intercostal blocks; effective clearing of the tracheobronchial tree by tracheal suction, bronchoscopy, or tracheostomy; and relieving mental apprehension is the therapy that applies to most lung injuries.

Traumatic pneumothorax may occur after simple rib or sternal fracture. In small nonprogressing pneumothorax, hemothorax, or combination thereof, observation or simple needle thoracentesis will suffice. If collapse is progressive, closed pleural space tube drainage should be done. Occasionally, if bleeding is from a systemic chest wall artery, thoracotomy and ligation are necessary.⁵⁻⁷

TRACHEOBRONCHIAL TREE

Injuries to the tracheobronchial tree are more common than realized. The shearing area of stress in deceleration is at the bifurcation of the trachea or the major bronchi from the trachea. Rupture may also occur from instinctive closing of the glottis as danger looms. The compression force literally "blows out" the weakest area of the tracheobronchial tree (table 1). Complete torsion of the lung without bronchial rupture has been diagnosed after injury by roentgenogram. Gangrene rapidly results if torsion is not reduced.

Diagnosis of rupture of the tracheobronchial tree is presumed in the presence of a chest injury with subcutaneous emphysema, persistent pneumothorax and atelectasis, mediastinal emphysema, and hemoptysis. However, if mediastinal soft tissue is interposed between the severed bronchial ends, the only finding clinically and by roentgenogram is a gradual atelectasis of the lung or lobe. Tracheal tears are usually more dramatic in symptoms and more easily diagnosed. Depending on the site of injury, dyspnea may be profound or minimal (see Case 1).

TABLE 1
RUPTURE OF TRACHEOBRONCHIAL TREE

1. Associated injuries in 84 cases	
a. No injuries	50 %
b. Fractures—thorax	31
pelvis	5.9
other	4.7
c. Head injuries	3.6
d. Esophageal rupture	2.4
e. Other	2.4
	100 %
2. Etiology of injuries	
a. Automobile collision	29.8%
b. Crushed (vehicle)	19.2
c. Run over (vehicle)	15.5
d. Vehicle (pedestrian)	8.3
e. Nonvehicle injury	27.2
	100 %

Surgical repair at the earliest opportunity offers the best result. Pulmonary resection is advisable if a satisfactory anastomosis cannot be done. Even years after injury, delayed anastomosis has been satisfactorily performed, but this is unusual, as the atelectatic lung is often "destroyed" by infection. If it is not destroyed anatomically, the lung may regain a portion of physiologic function.⁸⁻¹¹

AORTIC INJURY

The paucity of external visible chest damage is often amazing, considering the degree of aortic injury that may occur. Examination of pathologic specimens has revealed five degrees of aortic damage: (1) isolated or multiple intimal hemorrhage, usually of no clinical significance; (2) intimal laceration and subintimal hemorrhage; (3) internal laceration through intima and into media; (4) rupture through entire wall except adventitia; and (5) laceration of all layers with formation of mediastinal hematoma and potential false aneurysm.

This trauma may be localized or in multiple sites. Most commonly, significant injuries are either in the ascending aorta near its origin or just distal to the left subclavian artery—the isthmus area. Severe myocardial damage is usually found associated with the ascending arch laceration. The combination contributes to the high mortality of this injury (tables 2 and 3).

Sequellae of the various degrees of aortic trauma are thrombosis and embolization, dissecting aneurysm, delayed rupture, and false aneurysm formation.

TABLE 2
SITE OF RUPTURE OF AORTA

<i>Injury</i>	<i>Ascending</i>	<i>Arch</i>	<i>Isthmus</i>	<i>Thoracic</i>	<i>Abdominal</i>	<i>Multiple</i>	<i>Total</i>
Isolated	10 %	9.4	55.6	15.8	6.4	2.8	100%
Heart	45.1%	5.8	27.8	7.8	2	11.5	100%

TABLE 3
SURVIVAL TIME IN CLOSED HEART AND AORTIC INJURIES

	<i>DOA</i>	<i>0 to 6 hrs.</i>	<i>6 to 24 hrs.</i>	<i>1 to 4 days</i>	<i>5 to 50 days</i>	<i>2 to 10 mos.</i>	<i>1 to 4 yrs.</i>	<i>Cured</i>	<i>Total</i>
Aorta	86 %	3	1.5	1.8	4.7	1.1	1.1	0.8	100%
*Heart	86.8%	6.6	2.2	3.6	0.8	0	0	0	100%

*This represents a survey of autopsy records.

The majority of individuals suffering significant aortic injury die within a few minutes, either from aortic or other bodily trauma. About 15 per cent of these patients reach a medical installation and survive long enough for diagnosis and possible surgical repair. A patient rarely lives more than twelve months without surgical intervention. The most favorable site for repair, and also the most common area of injury with survival, is the isthmus of the aorta.

Mechanisms of aortic damage are (1) shearing action of the bony framework, as from a fracture or fracture-dislocation of a vertebra; (2) sudden compression of the abdominal aorta with a proximal "blow-out" from the volume of "trapped" blood in crush accidents; or (3) deceleration tissue strain at the junction of the relatively fixed aortic arch and the movable heart and thoracic aorta.

The most important aids in diagnosis are serial chest roentgenograms. The most consistent findings are (1) a widened mediastinum in the para-aortic portion; (2) fractures of the sternum, clavicles, upper rib cage, or dorsal spine; (3) early or delayed hemothorax; (4) unexplained shock; and (5) evidence of a rapidly advancing mediastinal mass—dyspnea, cyanosis, or dysphagia. These signs and symptoms may occur from hours to weeks after injury.

Surgical repair by primary suture or insertion of a prosthetic graft offers the only cure (see Case 2).^{1,12-14}

HEART AND GREAT VESSELS

Direct injuries to the heart probably occur in about 15 per cent of fatal, nonpenetrating chest traumas (table 3). The degree of heart injury is directly related to the intensity and duration of the forces. Pericardial effusion and lacerations, epicardial hemorrhage, and myocardial and endocardial changes indistinguishable from

myocardial coronary infarction have been found. Subendocardial damage occurs in most severe injuries, and damage is moderate in about 50 per cent. Ruptures of mitral valve leaflets, chordae tendineae, or intraventricular septum are not uncommon. Delayed myocardial rupture is a very real possibility.

The patient may demonstrate anginal pain, pericardial friction rub and effusion, changing murmurs, and an abnormal cardiac rhythm. Early or late cardiac decompensation is not unexpected. Traumatic coronary occlusion may suddenly develop. The electrocardiograph will record wide varieties of rhythm and changes in individual complexes. In the absence of a history or prior evidence of heart disease, such findings support a presumptive diagnosis of heart injury.

Treatment depends on the degree of cardiac involvement. Ruptured valves and resulting insufficiency may need surgical repair if the patient cannot be stabilized medically. The majority of cardiac injuries are treated conservatively. Cardiac tamponade may call for emergency measures.

Damage to great vessels other than the intercostal and internal mammary arteries is surprisingly uncommon. Bleeding from an internal mammary artery may simulate a rupture of the aorta clinically and on the posteroanterior chest roentgenogram. Lateral roentgenograms will aid in differentiation. Surgical ligation may be needed.¹⁵⁻¹⁷

DIAPHRAGM

Traumatic rupture is the second most common type of diaphragmatic defect in the adult. Only the esophageal hiatal hernia is more common. Except for unusual cases, trauma results in a defect of varying size in the *left* hemidiaphragm, usually in the tendinous portion but often in the

periphery or extending completely across the diaphragm into the esophageal hiatus. Evisceration of varying amounts of intraabdominal content enters the chest. Incarceration or strangulation results with accompanying symptoms of disturbed intestinal physiology.

Diagnosis depends on the degree of evisceration and the secondary alteration in pulmonary or intestinal physiology. Massive movement of abdominal contents into the chest is an emergency situation and usually is not too difficult to diagnose by roentgenographic and clinical findings.

Smaller incarcerations are often not diagnosed without roentgenologic help. Shoulder-strap muscle pain is usually masked by other associated injuries. Bowel obstruction may not occur until weeks or even years later. During the interval, the individual may have vague symptoms suggestive of biliary tract obstruction, partial bowel obstruction, peptic ulcer, or coronary disease.

Roentgenograms of the upright chest demonstrating a "high diaphragm," fluid-air levels in the thoracic cavity, shift of the heart to the right, and fluid in the pleural space are most suggestive of a ruptured diaphragm (see Case 3).

Usually a compression force applied to the

lower chest or upper abdomen forces against the negative intrathoracic pressure and literally rips the seams of the diaphragm. On the right, the same force is distributed more evenly by the bulk of the liver. Injuries to intraabdominal viscera, usually the spleen, are often associated.

Surgical repair at the most favorable time for the welfare of the patient is the therapy of choice.¹⁸⁻²⁰

CHYLOTHORAX

Chylothorax is a rare sequella of injury. Symptoms and signs are dependent on the amount and rapidity of collection of chyle within the pleural cavity. Conservative therapy by thoracentesis is usually advocated for a period of ten to fourteen days. If the duct does not close spontaneously, closed chest tube drainage for two to three days should be tried. If this is unsuccessful, open thoracotomy and ligation of the duct are necessary.

The major problem is not infection, as chyle is bacteriostatic. Rather, the high protein and fat loss in the removed chyle makes oral replacement extremely difficult; the patient may rapidly fail, due to severe nutritional deficits. Reexpansion of the lung may also be a difficulty, as in any prolonged collapse and fibrous peel forma-

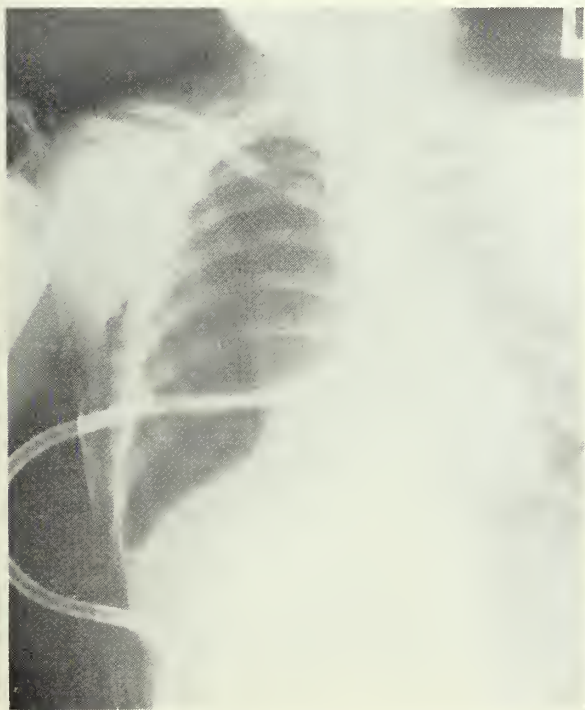


Fig. 1. Case 1. Chest roentgenogram shows pneumothorax, shift of mediastinum, and subcutaneous emphysema.



Fig. 2. Case 1. Following thoracotomy, improvement is evident on x-ray.

tion. The ribs are often intact, and discomfort is minimal except for dyspnea.²¹

ESOPHAGUS

Esophageal injury is another infrequent result of closed chest trauma. Several cases of tracheo-esophageal fistula have occurred. Signs and symptoms are related to foreign body contamination of the tracheal bronchial tree from swallowed saliva, regurgitant gastric juice, or food. These symptoms may occur a few days after injury. Recurrent pneumonitis may be the most persistent finding.

Diagnosis is aided by endoscopy of the tracheobronchial tree and esophagus. Instillation of iodide contrast material in both organs may further delineate the problem. Barium contrast methods are contraindicated.

Treatment consists of surgical division and closure of the fistula. Adequate time preoperatively should be given to restore the patient to positive nitrogen balance, to reduce the inflammation around the fistula to a minimum, and to allow all pneumonitis to clear or stabilize.^{22,23}

CASE REPORTS

Case 1. S.R., an 8-year-old white girl, was run over by an automobile. She was admitted to the hospital with a right tension pneumothorax causing severe respiratory distress. There were no rib fractures. Emergency thoracentesis alleviated dyspnea partially, but the pneumothorax persisted. Despite closed pleural space tube drainage and constant egress of large amounts of air, pneumothorax, shift of the mediastinum, and subcutaneous emphysema persisted (figure 1). The presumptive diagnosis of ruptured bronchus was confirmed at thoracotomy forty-eight hours after injury. Complete disruption of the right intermediate bronchus at its bifurcation into the middle and lower lobe bronchi had occurred. Resection of the involved lobes resulted in a cure and no demonstrable pulmonary symptoms. The right upper lobe expanded well to fill the space, and, except for mediastinal pleural thickening and elevation of the right hemidiaphragm, the chest x-ray appeared excellent (figure 2).

Case 2. D.S., a 22-year-old white man, was an automobile passenger who was critically injured in a two-car collision in 1956. He suffered head and right chest cage injury. After a critical period of several weeks, full recovery apparently resulted. In the latter months of 1959, the patient consulted his physician again because of vague left chest distress. A roentgenogram disclosed a large aneurysm of the thoracic aorta distal to the left subclavian artery (figure 3). Review of the original films disclosed widening of the mediastinum and left pleural fluid but no pneumothorax. Gradual resorption of pleural fluid occurred, but the widened mediastinum resolved only in part. The aortic aneurysm soon became evident. The patient was referred for treatment.

The clinical examination was not remarkable. There was no bruit over the posterior chest and the peripheral pulses were equal, as was the extremity blood pressure. Diagnosis of a false aneurysm of the aortic isthmus was confirmed at surgery. The aneurysm had invaded the

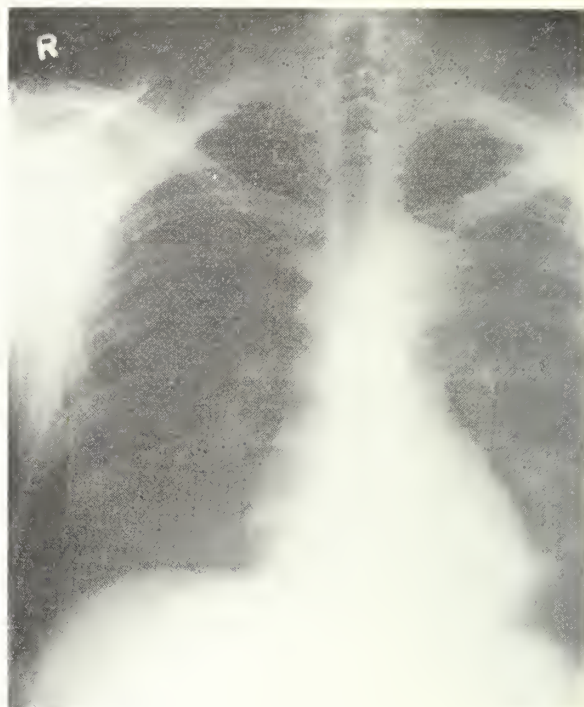


Fig. 3. *Case 2.* Large aneurysm of thoracic aorta is seen distal to the left subclavian artery.



Fig. 4. *Case 2.* Postoperative roentgenogram shows normal aorta following resection and end-to-end anastomosis.

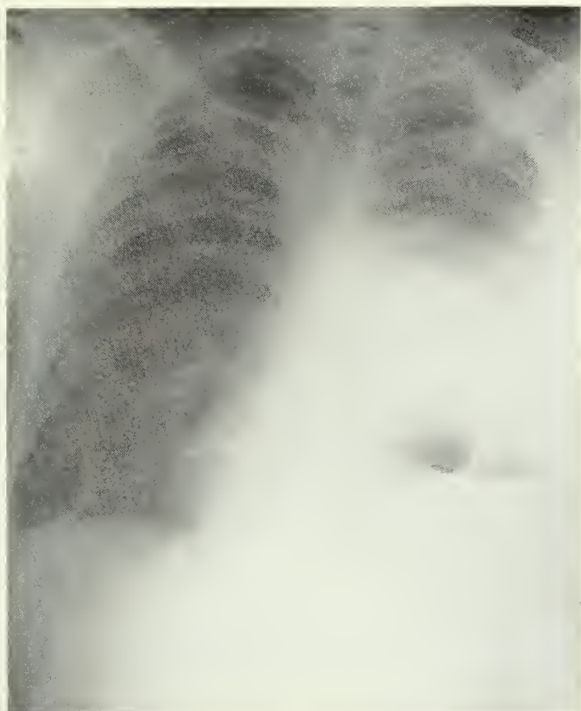


Fig. 5. *Case 3.* Shift of heart to right is seen with fluid and compression changes in lower lung field.

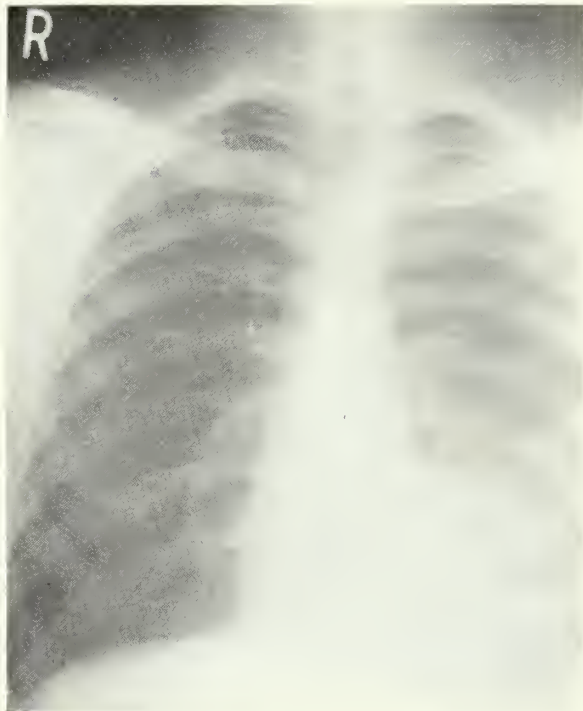


Fig. 6. *Case 3.* Only pleural changes and high left diaphragm are noted following double-barrel colostomy, resection of involved colon, and diaphragmatic repair.

mediastinal structures. Resection and end-to-end anastomosis was possible. The aorta appeared normal on the postoperative films and has remained so to date (figure 4).

Case 3. J.A., a 35-year-old intoxicated man, drove his automobile into a telephone pole. He was examined in a hospital emergency ward and told that a "couple of ribs" on the left were fractured from the steering wheel, but no roentgenograms were taken. The patient was sent home. In the next forty-eight hours, left shoulder-strap pain, vomiting, and fever developed. He was admitted critically ill to the county hospital late in the afternoon as "a pneumonia case." Thoracic surgery consultation was requested to treat the "empyema." However, the chest roentgenogram showed shift of the heart to the right, fluid in the left pleural space, an air-fluid level probably above the diaphragm, and compression changes in the lower lung field (figure 5). After suitable therapy, emergency thoracotomy disclosed an incarcerated gangrenous loop of colon protruding through a 6-cm. tear in the dome of the left diaphragm. A double-barrel colostomy, resection of the involved colon, and diaphragmatic repair resulted in eventual cure. A chest roentgenogram revealed only pleural changes and a high left diaphragm (figure 6). X-rays presented through courtesy of C. Dotter, M.D.

DISCUSSION

The major point of emphasis is that closed thoracic trauma may cause serious anatomic and physiologic wounds of intrathoracic viscera, with only a very minor degree of visible evidence. This is the deadly trap for the patient and physician.

Primary shock from thoracic organ damage is too often falsely attributed to the more dramatic appearing head and extremity traumas. Isolated chest accidents should not be dismissed without careful evaluation. Latent serious complications may erupt and result in prolonged morbidity or death.

Alertness to these possibilities is the physician's best stimulus to diagnosis. Often it may be the patient's only chance for survival.

SUMMARY

A description of the etiologic forces and the various types of visceral damage from nonpenetrating chest trauma has been outlined. Visceral injury is often significant despite minimal external evidence of violence. Serial chest roentgenograms and electrocardiograms are the most useful laboratory aids in diagnosis. Alertness of the physician is the key to the diagnosis and treatment.

Case 1 was presented through the courtesy of Lawrence Lowell, M.D., thoracic surgeon in Portland, Oregon.

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PEPTIC ULCERATION may be a common complication of acute traumatic as well as chronic respiratory insufficiency. Therefore, it is suggested that prophylactic ulcer therapy be provided for patients with flail chest—multiple rib fractures, paradoxical respiratory movements, and, usually, respiratory insufficiency. Massive hematemesis beginning five and seventeen days after injury was fatal in 2 such patients. In a third patient, previously treated for duodenal ulcer, bleeding contributed to death. Carbon dioxide retention accompanied extreme pulmonary insufficiency in all 3 patients. Gastrointestinal hemorrhage has not occurred during treatment for flail chest since use of antacids was made routine.

P. P. SALTER, JR., and C. LYONS: Gastric hemorrhage as a complication of flail chest. *Am. J. Gastroenterol.* 32:500-508, 1959.

Neonatal Intestinal Obstruction

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THE PAST SIX decades have witnessed remarkable improvement in salvage of babies with intestinal obstruction.¹⁻⁴ In centers in which such problems are faced daily, mortality rates improve constantly. In hospitals where only an occasional case is seen, the baby still sometimes suffers. Either an enthusiastic staff outdoes itself with diagnostic study, or the gravity of the situation is not appreciated. In either case, effective treatment lags. For this reason, certain points in management of neonatal intestinal obstruction warrant reiteration.

This paper presents pertinent data in a group of hospitalized infants, 8 with *upper* intestinal obstruction and 2 with lower bowel blockage. These cases were selected from a larger series of such hospitalized cases, numbering almost 100, to illustrate certain facts relative to surgical management of infant intestinal obstruction. The following material attempts to show the diagnostic value of plain flat and upright roentgenograms of the intestinally obstructed newborn. Emphasis is placed on *avoidance* of the use of barium in these critically ill infants, except as a last diagnostic resort. Attention is called to the type and degree of metabolic derangement which can be expected to accompany upper intestinal obstructions, and why. In such cases, with clinical time at a premium, only the most essential laboratory studies are indicated; these are discussed. The importance of swift and precise restorative measures is stressed. By adherence to such details of preoperative management, the surgeon can enter into what is often a technically difficult procedure, safe in the knowledge that the best current principles have been applied.

CASE REPORTS

Case 1. A 12-day-old, 2,900-gm. Negro male had been vomiting for six days. For four days, he had had no stool, and urine output had become progressively scanty. On admission, there was marked dehydration, and tetany was present. The roentgenogram showed duodenal obstruction. The metabolic picture was one of hypochlo-

remic alkalosis. Parenteral therapy for tetany and dehydration was pursued vigorously for twenty-seven hours prior to surgery. At operation, a Ladd procedure for incomplete rotation of the colon was performed.⁵ The infant was discharged three weeks after admission, at which time birth weight had been regained (figure 1).

Case 2. L.C., a 49-day-old white female, weighed only 2,900 gm. on admission. There had been some regurgitation at times since birth, which had become acute in the two weeks preceding admission and apparently was bile-free. The barium roentgenogram was interpreted as showing an obstruction of the pylorus. No clear cut "string sign" was noted, however. At laparotomy, no hypertrophy of the pylorus was found, and the abdomen was closed. However, signs of upper intestinal obstruction continued, the characteristic metabolic picture was noted, and tetany developed. After vigorous parenteral treatment, the infant was reexplored four days later, and a septal stenosis of the duodenum was finally located. A duodenoduodenostomy was performed, but the infant succumbed several days after this procedure. At autopsy, peritonitis and a large intraventricular septal defect were found (figure 2).

Case 3. R.A.S. was a 3-day-old, 2,200-gm. white female. She had vomited most feedings since birth, and a 500 gm. loss of weight occurred. Plain flat film, without contrast media, gave evidence of obstruction, probably in the second portion of the duodenum. Chemistries revealed hypochloremic alkalosis. Surgery was withheld until parenteral therapy had directed electrolyte levels toward normal. At operation, the infant was found to have atresia of the duodenum proximal to the common duct, a "ring" pancreas, an incompletely rotated cecum, and a Meckel's diverticulum. A duodenojejunostomy was performed, and the infant passed stool on the second day (figure 3).

Case 4. S.W., a 3,000-gm., first-born white male, was admitted when he was 37 days old. History was characteristic of hypertrophic pyloric stenosis, and a confirmatory palpable mass was found. After correction of the metabolic abnormalities shown, a Ramstedt operation was performed (figure 4).

Case 5. T.C., a 2,100-gm. male, had vomited all feedings during the three days of life. Vomitus contained bile. The roentgenogram showed several dilated loops of small intestine, indicating a somewhat lower block than the preceding cases. Again, chlorides were depressed and CO₂ was elevated. A cutdown was performed, and fluids were given for proper correction. Laparotomy revealed an atresia of the midjejunum. The area was resected, and jejunojunctionostomy was performed. The infant was promptly relieved (figure 5).

Case 6. A 23-day-old boy, weighing 3,200 gm., was admitted after four days of severe bile-free vomiting. Mod-

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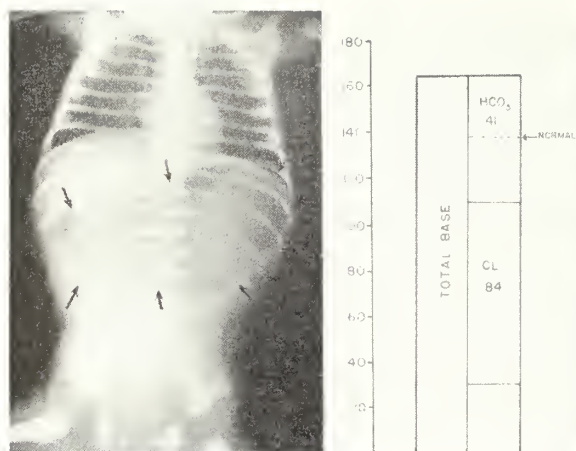


Fig. 1. Incomplete rotation of colon in 12-day-old male. Note tremendous distention of stomach and lack of gas in lower bowel. Metabolic picture was that of severe hypochloremic alkalosis.

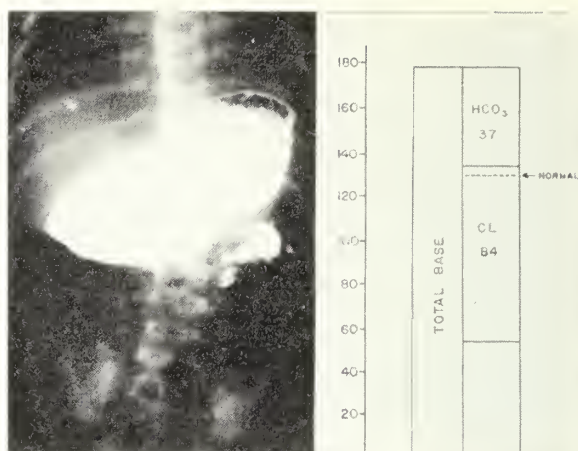


Fig. 4. Distended stomach from hypertrophic pyloric stenosis outlined by barium (usually not necessary). Characteristic hypochloremic alkalosis was present.

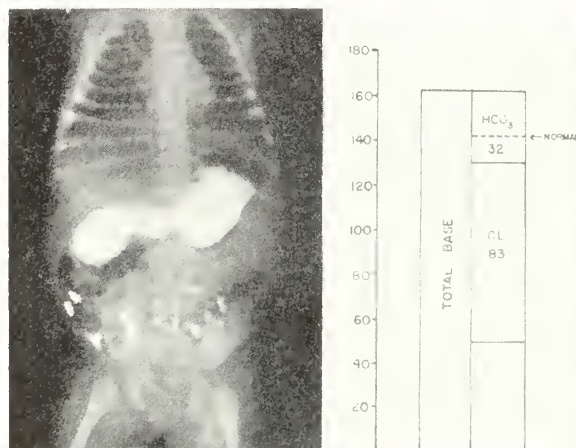


Fig. 2. Obstruction caused by septum across second portion of duodenum with only pinpoint perforation, which did allow dribblets of opaque media to pass. Hypochloremic alkalosis developed.

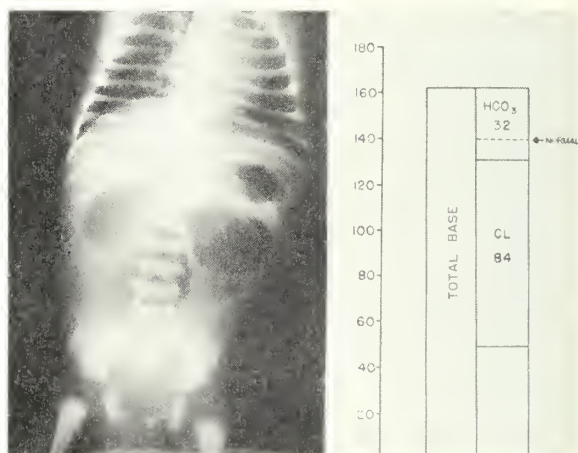


Fig. 5. Distended loops of upper intestine in case of high jejunal atresia. Hypochloremic alkalosis was moderate.

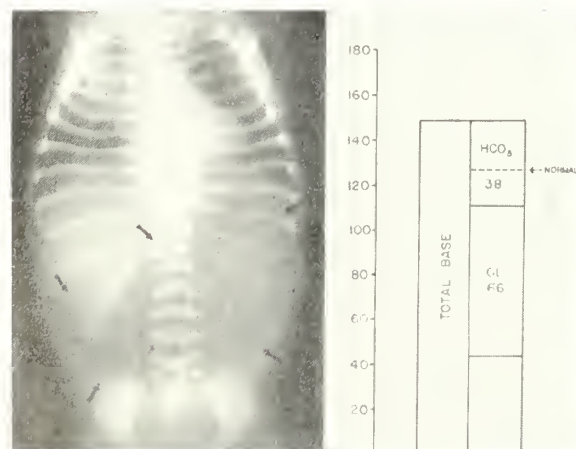


Fig. 3. Marked distention of stomach due to annular pancreas with duodenal atresia. Meckel's diverticulum and incomplete rotation of colon were also found at surgery. Hypochloremic alkalosis was pronounced.

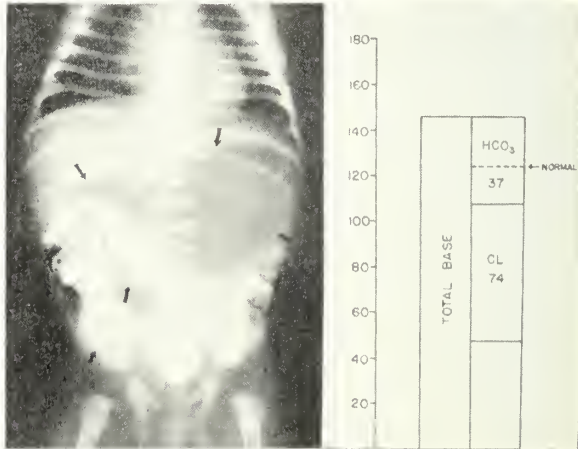


Fig. 6. Distention of stomach secondary to hypertrophic pyloric stenosis outlined by arrows. Condition was accompanied by hypochloremic alkalosis. Barium enema ruled out incomplete rotation of colon.

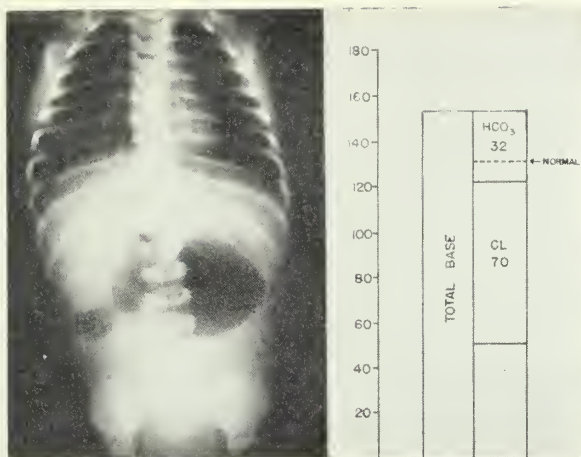


Fig. 7. Air pattern defines another duodenal atresia in 3-day-old. Note hypochloremic alkalosis.

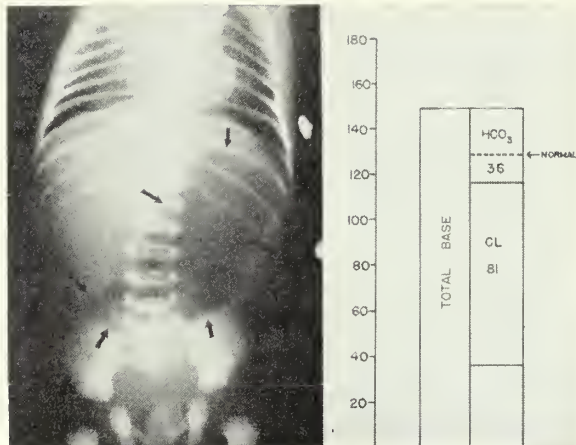


Fig. 8. A 4-day-old infant with duodenal atresia in whom metabolic alkalosis with hypochloremia also developed.

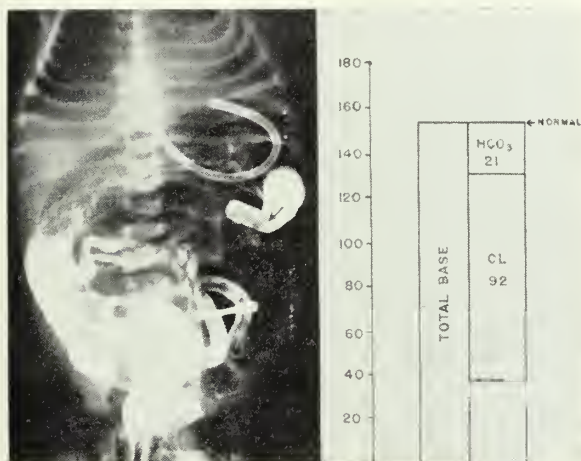


Fig. 9. In contrast to preceding 8 cases, a low intestinal blockage was *not* accompanied by hypochloremic alkalosis. This case of meconium plug of descending colon is outlined by a thin barium enema. Plug extended to point marked by arrow. Above this, colon is distended.

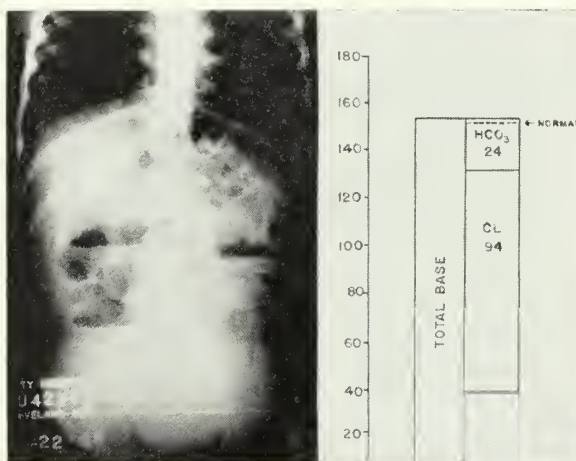


Fig. 10. Again, blocking of ileum was *not* accompanied by hypochloremic alkalosis. Multiple fluid levels were due to a volvulus of midgut about a vitelline duct remnant.

erately severe dehydration was present, and the electrolyte picture as shown was found (figure 6). A palpable pyloric tumor was detected by several examiners. Infant was prepared for surgery with intravenous sodium chloride plus potassium.⁶ A Ramstedt pyloromyotomy was then performed, which produced satisfactory relief of the pyloric stenosis. The baby was discharged from the hospital after one week.

Case 7. G.O., a 3-day-old white female, developed projectile vomiting on the second day of life. When seen twenty-four hours later, the distended stomach almost completely filled the abdomen (figure 7). The accompanying hypochloremic alkalosis was corrected, and a side-tracking duodenojejunostomy was performed. The baby survived.

Case 8. Another duodenal atresia patient, J.P., was a 2,300-gm. Negro male, who was subjected to duodenojejunostomy after correction of dehydration, hypochloremia, and alkalosis. Age was 4 days, and vomiting had

continued for forty-eight hours. Recovery was complete in one month (figure 8).

Case 9. L.K., a 2,300-gm. white male, became progressively distended to the fourth day of life. Roentgenograms showed distention of both small and large bowels down to the splenic flexure. Flushing enemas proved of no avail, and a transverse colostomy was performed. A meconium plug was indicted.⁷ Biopsy of colon revealed the presence of normal ganglion cells. In this newborn, chemistries were in contrast to the preceding 8 cases: chloride was 92, and carbon dioxide was 21. The colostomy was successfully closed at 3 months (figure 9).

Case 10. A 20-month-old Negro female was admitted in an extremely serious condition with severe dehydration and shock. In spite of a urinary specific gravity of 1.035, chloride was 94, and CO_2 , 24. After restoration, laparotomy revealed a midgut volvulus about a vitelline duct remnant. Several centimeters of gangrenous ileum had to be resected. Recovery was prompt (figure 10).

DIAGNOSIS	PATIENT	RACE	SEX	AGE AT OPERATION	DURATION OF VOMITING	DEGREE OF OBSTRUCTION	URINE SPEC. GRAVITY	HGB CONCENTRATION	MEQ CARBON DIOXIDE	MEQ CHLORIDES	MEQ SODIUM	MEQ POTASSIUM	SURGICAL TECHNIQUE	OUTCOME	DAYS IN HOSPITAL
1 MALROTATION	1	M	M	12	6	M	1035	19	41	84	150	65	LADD	S	21
2 DUODENAL STENOSIS	2	M	M	49	14	M	1028	17	32	83	151	3.9	1 LAP 2 DUO-DUO	D	17
3 ANNULAR PANCREAS	3	M	H	3	2	H	1026	16	38	66	134	45	DUO-JEJ	S	30
4 PYLORIC STENOSIS	4	M	M	37	14	M	1030	18	37	84	155	51	RAMSTEDT	S	10
5 JEJUNAL ATRESIA	5	M	H	3	2	H	1028	20	32	84	150	51	JEJ-JEJ	S	32
6 PYLORIC STENOSIS	6	M	L	23	4	L	1027	16	37	74	130	4.0	RAMSTEDT	S	8
7 DUODENAL ATRESIA	7	M	H	3	1	H	1025	19	32	70	141	4.7	DUO-JEJ	S	30
8 DUODENAL ATRESIA	8	M	H	4	2	H	1026	18	36	81	135	4.4	DUO-JEJ	S	35
9 MECONIUM PLUG SYNDROME	9	M	L	4	2	L	1030	17	21	92	145	5.6	COLOST.	S	42
10 ILEAL VOLVULUS	10	M	L	600	5	L	1035	9	24	94	130	5.1	RESECT	S	14

Table 1. "Gamblegram" representing, on the left, electrolytes of extracellular space in normal babies of this age. On the right is shown the average deviation from normal as found in the first 8 cases herein presented.

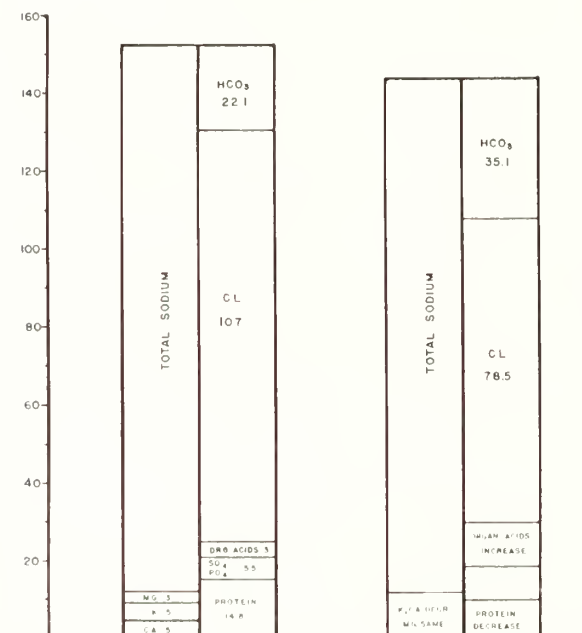


Table 2. Summary of pertinent data in this series of infants. Within the heavy dark line are included the most important data.

DISCUSSION

Table 1 is a "Gamblegram" contrasting the electrolyte picture of the extracellular space in the normal baby of this age with the *average* deviation of those infants in this series having upper intestinal obstruction. Table 2 summarizes this series. Within the heavy dark line may be observed the urine specific gravities and the hemo-

globin concentrations, indications of the degree of dehydration present in most cases. Whereas the carbon dioxide and chloride determinations were deviated in a consistent direction, determinations of sodium and potassium showed rather scattered findings.

That hypochloremia and alkalosis result from obstruction of the pylorus, duodenum, and upper jejunum was amply demonstrated several years ago in the experimental animals of Elman and Hartmann, Hadden and Orr, McIver, and a number of others,⁸⁻¹⁰ who showed that animals with simple ligature obstruction of the upper intestine developed lowered chlorides and increased alkali reserves, leading ultimately and, in fact, rather promptly, to death. They also showed that administration of salt solution would lengthen such animals' lives a number of weeks. Dogs given sodium bicarbonate solution, on the other hand, died just as soon, or sooner, than those left untreated.

Table 3 diagrams the mechanism of this metabolic alkalosis. Anything which removes a large amount of chloride from the stomach, be it vomiting or tube drainage, reduces the plasma electrolyte level of this ion. Increased plasma bicarbonate compensates in an attempt to maintain ionic equilibrium. If excessive loss is continued, however, buffering mechanisms prove inadequate, and plasma pH increases. There is also an extracellular dehydration secondary to unreplaced gastric and renal loss of water, and this extracellular fluid loss is superimposed on the extracellular chloride deficit. Furthermore, when alkalemia persists for any length of time, a diminution in the ionized fraction of calcium

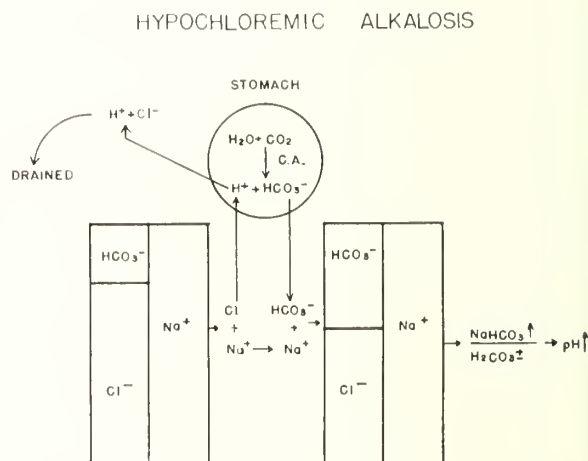


Table 3. Graphic representation of the mechanism of development of metabolic hypochloremic alkalosis and alkalemia.

develops. As a consequence of starvation, plasma proteins become reduced. Development of tetany depends upon the relation of the ionized calcium to the albumin fraction of protein.¹¹ Therefore, tetany in these cases may be encountered even in the face of normal serum calcium levels.

Besides illustrating metabolic aberrations of neonatal intestinal obstruction, these cases serve to demonstrate several important features in management. Simple flat abdominal films, which usually include the entire body, *without* contrast media, are often most informative and shorten the time before which definitive therapy can be undertaken. When accompanied by an upright view, much information can be gained.

In dealing with suspected upper intestinal obstruction, blood chemistries should be obtained *early*. The finding of hypochloremic alkalosis adds weight to a presumptive diagnosis of high obstruction. The degree of deviation of chemistries from normal varies directly with the *degree* of obstruction and with its *duration*. Acceptable modern operative preparation *demand*s restoration of fluid and electrolytes to normal or near normal levels. Only then can general anesthetics be administered safely. The surgical techniques necessary for correction of most of these anomalies require use of general, rather than local, anesthetics.

Treatment of these small patients must be safe, swift, and sure. History of emesis in the neonate should include details on amount, type, and duration. Bile-free, projectile vomiting of recently ingested feeding points to a high level of obstruction. Physical examination must include note of baby's general condition, turgor of skin, condition of fontanelles and eyeballs, and close observation of abdomen. Simple flat and upright abdominal films are most revealing at this age and often preclude the need for exhausting, time-consuming barium studies.

As these case records indicate, blood chemistries are rather characteristically altered, depending upon duration of condition. When blood specimens are at a premium, it is important to determine carbon dioxide and chloride levels as well as the hematocrit and specific gravity of urine, which provide further information as to the degree of hydration. Where blood pH can be measured directly, less guess work is involved.

Once roentgenograms have been taken and blood and urine specimens sent for analysis, immediate steps must be directed toward restoration of the normal state. These include decompression of the overdistended stomach and re-

hydration of the depleted extracellular compartment. In cases involving shock, blood replacement and oxygen by catheter are indicated.

Effective gastric decompression is potentiated by use of a small plastic urinary catheter placed transnasally. Perforations at the tip are enlarged and increased with scissors before placement in the stomach. After positioning, this tube is irrigated with frequent small amounts of normal saline. Constant suction through these small catheters is much more effective when this plan is followed.

Adequate rehydration is likewise virtually impossible without use of a small vinyl catheter in a saphenous vein. Properly conducted, such a "cutdown" procedure can be accomplished in less time and has proved more dependable by far than use of scalp veins in tiny infants. *Clysis administration of fluids should be abandoned*.¹² Calculation of volume and types of replacement solutions are dependent upon laboratory findings. There are more ways to determine these dosages in print at the present time than there are conditions to treat. The reader is referred to any of the several excellent discussions of this subject listed in the bibliography.^{11,13-17} In addition, almost all pharmaceutical companies manufacturing replacement solutions provide tables, charts, and pocket calculators.

Decision as to the appropriate time for and type of surgery needed varies, depending on the individual. Here the true "art of medicine" manifests itself. There are those who prefer local and those who insist upon general anesthesia. Some depend upon side-to-side anastomoses, whereas others demand end-to-end suturing. Whatever the personal penchant, a few requirements are indispensable through all this type of surgery. Gentleness and precision are *sine qua non*;¹⁸ attention to temperature regulation is no less vital. Once the operation is over, constant postoperative vigilance is mandatory.

If these practices, developed in hospitals where such problems are faced daily and where further refinement of techniques are continually undergoing revision, are consistently applied, more and more infants will survive urgent surgery.

CONCLUSION

During the decade from 1890 to 1900, the surgical treatment of congenital pyloric stenosis was attended by a mortality rate of 60 per cent. In the present decade, rates of less than 1 per cent are consistently reported.

On the other hand, Evans, in an exhaustive review of 4,100 articles to 1950, states that atresias

of the gastrointestinal tract carry a 76 per cent mortality rate.¹⁹ If this be the case, much room for improvement of clinical care remains.

Pertinent laboratory data and clinical findings in a group of infants having upper intestinal obstructions of various types have been reviewed. In contrast to others with lower obstructions, these infants demonstrated rather consistent development of hypochloremic alkalosis. The occurrence of this metabolic derangement in such cases is to be expected. Attempts at experimental duplication of these lesions in laboratory animals produce a similar metabolic picture.

Severity of metabolic derangement depends upon degree of obstruction and its duration. Appreciable deviations from the metabolic norm deserve specific therapy in preparation for surgery. Techniques found effective in such management are described. After appropriate corrective steps have been taken, the surgeon must rely upon his instinct to judge the proper time for operative intervention. Avoidance of unnecessarily time-consuming studies results in higher survival rates.

Acknowledgment is made to those physicians who graciously permitted this survey of their hospital charts.

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CARCINOMA of the extrahepatic biliary tract is slow-growing and usually incurable by the time of diagnosis. However, surgical intervention may provide significant palliation and prolong life.

Tumors located high in the biliary tract can rarely be removed, and even palliation is difficult. For lower lesions, resection is advisable if the operative risk is not too great. If little alternative is offered, resection for cure should be attempted even when risk is great. If the lesion cannot be removed, short-circuiting operations can be performed. Other decompression techniques are used only when more radical procedures are impossible.

Among 20 patients with tumors, satisfactory decompression of the biliary tract was effected in 18, with only 1 operative death. In 2 patients with carcinomas of the right and left hepatic ducts high in the hilus, decompression was not attempted. Seven of the patients have survived for periods ranging from six months to seven years: 5 of 9 after radical pancreaticoduodenectomy, 1 of 4 treated by simple catheter drainage, and 1 after biliary-enteric anastomosis with local resection.

H. N. LIPPMAN, L. C. McDONALD, and W. P. LONGMIRE: Carcinoma of extrahepatic bile ducts. *Ann. Surg.* 25:819-826, 1959.

Current Concepts of the Cervical Portion of the Sympathetic Nervous System

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THE AUTONOMIC, or involuntary, nervous system consists of two divisions: the sympathetic and the parasympathetic. Since Langley¹ and Gaskell,² it has been held that the cells of origin of preganglionic sympathetic neurons are found only in T1 to L2 segments of the spinal cord. Laruelle,³ however, has demonstrated the presence of sympathetic cell bodies also in the cervical portion of the spinal cord, where they are located in the mediolateral gray matter at the base of the anterior horns at levels C4, C5, C6, C7, and C8 (figure 1).

The preganglionic sympathetic fibers, together with the somatic motor nerve fibers, leave the spinal cord in ventral roots C5, C6, C7, C8, and T1. They reach the periphery by way of sympathetic nerves, periarterial plexuses, and somatic nerves. On emerging from the spinal cord via the ventral roots, one part of the preganglionic neurons forms a synaptic connection with postganglionic neurons in the small ganglia of a deep sympathetic chain (figures 2 and 3), which has been discovered by Delmas and associates.⁴ The other preganglionic neurons simply traverse the deep chain of small sympathetic ganglia situated in the transverse canal and join the vertebral nerve, with which they reach the cervicothoracic group.

The deep chain consists of a tangled web of sympathetic fibers and of 2 to 5 small but macroscopically visible ganglia. It begins below the transverse canal in which it ascends along the posterior aspect of the vertebral artery from C7 to C4. The peripheral relay station of the vasomotor sympathetic outflow, the deep chain represents the continuation of the dorsolumbar laterovertbral ganglionated trunk into the neck.⁵ In the neck, it gives rise to postganglionic fibers which mediate vasoconstriction and travel to the brachial soma.

In addition to the deep sympathetic chain and

the classical ganglionated trunk, Guerrier⁶ recognizes in the neck a superficial collateral sympathetic chain (figure 3), the branches of which proceed to the cervical soma and the ganglia of which are visible only in the fetus.

The classical sympathetic trunk in the neck consists of superior, middle, and inferior ganglia connected by intervening cords. The inferior cervical ganglion is often united with the first thoracic ganglion, thus creating the stellate ganglion. The ganglia correspond to visceral ganglia. The efferent rami proceed to the viscera in the neck and the chest; the afferent branches form the vertebral nerve.

According to Tinel⁷ (figure 4), the fifth cervical spinal root carries sympathetic fibers which join

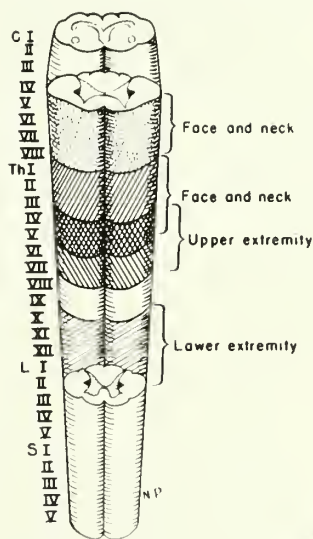


Fig. 1. Schematic drawing showing arrangement of the sympathetic gray matter in the spinal cord.

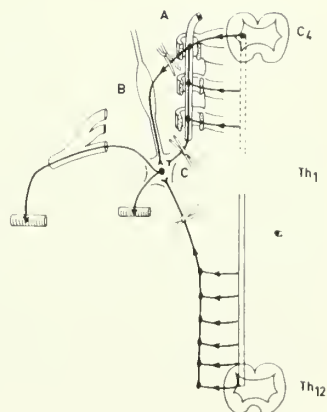


Fig. 2. Schematic drawing illustrating new concepts of the cervical group of sympathetic structures and pathways. The sympathetics, which control the arteries of the upper extremities, are emphasized. (Modified after ALLIEN).

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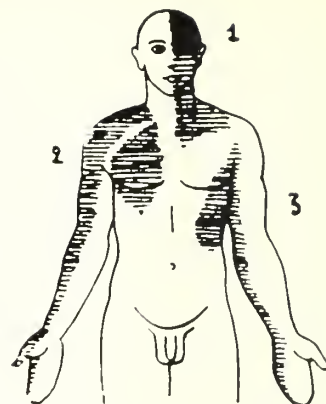
Fig. 3. Section through the lower cervical region, presenting the disposition of the three cervical sympathetic chains (*a*, *b*, *c*) and the vertebral nerves of Gnerrier.^{1,2} (*a*) Deep sympathetic chain. (*b*) Superficial collateral sympathetic chain. (*c*) Classic sympathetic trunk. (Courtesy of YVES GUERRIER).

the carotid plexus, furnishing, via the carotid and vertebral arteries, sympathetic innervation to the arteries of the head and the neck (cephalic sympathetic distribution). The sixth cervical root contains sympathetic fibers which proceed to the subclavian artery and the brachial plexus (brachial sympathetic distribution). The sympathetic fibers in the seventh cervical root are connected with the cardioaortic plexus, the thoracic branches of the subclavian and axillary arteries, and the phrenic nerves (thoracic sympathetic distribution).

Newer knowledge on the anatomy of the cervical portion of the sympathetic nervous system explains why its involvement by any one of a great variety of causes, for example, ventral rhizopathy provoked by discovertebral disease, may engender preganglionic stimulation or overactivity. This, in turn, may induce vasoconstriction or active vasodilation in certain arterial areas, with resultant headaches and craniofacial pains (sympathalgia and atypical facial neuralgia); dysesthesias and vascular pains in the upper extremities; and, finally, pains in the upper part of the back and in the anterior chest wall simulating angina pectoris. In contrast, implication of the somatic sensory and motor nerve

Fig. 4. Topography of cervical sympathetic root syndromes:

(1) sternocervicofacial type, (2) pectoro-scapulosuperior brachial type, and (3) costoaxilloinferior brachial type. (JULES TINEL. Courtesy of Masson & Cie, Paris).



fibers produce symptoms and signs solely in their own areas of distribution.

Other clinical manifestations due to stimulation of the cervical portion of the sympathetic nervous system include otoneuroophthalmologic phenomena like those occurring in the syndrome of Barré-Licou;⁸ lingual, pharyngeal, and laryngeal paresthesias;⁸ vertigo; the shoulder-hand syndrome;^{9,10} acroparesthesia; epicondylitis, radial styloiditis, trigger finger, and Dupuytren's contracture; and functional and organic heart disease.¹¹

SUMMARY

Newer anatomic findings pertaining to the cervical portion of the sympathetic nervous system are briefly reviewed.

Attention is called to the fact that, besides the thoracolumbar, there is a cervical source of preganglionic sympathetic supply.

The anatomic findings explain the topography of the clinical manifestations and syndromes of cervicosympathetic origin.

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Selection of Patients with Congenital Heart Disease for Cardiac Surgery

Office Procedures and When To Advise Ancillary Studies

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THE PHYSICIAN PRESENTED with a case of congenital heart disease today has to consider the possibility that his patient may be cured or greatly helped by cardiac surgery. He must be familiar with the operative possibilities and with the special investigative procedures that may be necessary in order to evaluate properly the diagnosis and the severity of the lesion. It is of paramount importance to realize that current understanding of the physical signs manifested by heart disease should enable the physician to make a preliminary diagnosis and selection of most cases for surgery without immediate recourse to such tests as right or left heart catheterization or angiocardiology. To be sure, these tests may be indicated for confirmation of diagnosis in even the most straightforward cases, but their availability should not seduce the physician from the great possibilities of physical diagnosis of heart lesions.

OPERABLE LESIONS

This list includes the more common conditions which may be cured or relieved by surgery, including open-heart surgery using the heart-lung machine: (1) patent ductus arteriosus (PDA); (2) ventricular septal defect (VSD); (3) atrial septal defect (ASD), both ostium secundum and ostium primum; (4) anomalous pulmonary venous drainage into the right atrium or superior or inferior vena cava, commonly associated with ASD; (5) valvular and infundibular pulmonary stenosis; (6) tetralogy of Fallot and its variants; (7) coarctation of the aorta; and (8) aortic stenosis.

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CONSIDERATION OF INDIVIDUAL LESIONS

Patent ductus arteriosus. All patients with uncomplicated patent ductus arteriosus should be cured by surgery. The optimum age for operation is probably between 4 and 12 years of age, but certainly younger and older patients may be satisfactorily operated upon. The ligation and division of a patent ductus arteriosus will restore the circulation to normal, obviate the likelihood of bacterial endocarditis, and probably prolong the patient's life.

When the typical murmur in the typical site is present, further investigation beyond the electrocardiogram and x-ray is usually unnecessary. However, when the murmur is atypical in character or site, particularly the latter, it is mandatory to consider other causes of a continuous murmur and to advise right heart catheterization and, if necessary, retrograde aortography. The usual murmur of the patent ductus arteriosus is a continuous systolic diastolic murmur, sometimes described as millwheel or machinery in character, with its maximal intensity around the time of the second heart sound. It is usually loudest at and above the pulmonary area. Beware of the continuous murmur, maximal at the third and fourth left interspace. Such a murmur is found in aneurysm of the aortic sinus which has perforated into the right ventricle or right atrium. Although this is a rare lesion, the writer has now seen 6 cases, 2 of which have been operated on for suspected patent ductus arteriosus. Among the lesions giving rise to a continuous murmur, which may be confused with patent ductus arteriosus, are aneurysm of the aortic sinus which has perforated into the right heart, arteriovenous fistula in the left lung, traumatic intrathoracic or intercostal systemic arteriovenous fistula, aortopulmonary septal defect, and the venous hum of young children. The latter

is probably the most common cause of a continuous murmur. It is heard over the left side of the neck and in the left upper chest and is due to a rapid flow of blood from the jugular system into the chest. Compression of the neck veins and downward head position will cause this murmur to disappear.

In addition to the true continuous murmurs, aortic stenosis and insufficiency and the rather rare condition of ventricular septal defect with aortic insufficiency may give rise to to-and-fro murmurs, which may be misinterpreted as continuous and therefore be confused with patent ductus arteriosus. Generally, the most important feature of these cases is the atypical character and site of the murmur. This in itself is an indication for further investigation.

During the first year of life, the continuous murmur may be absent. In the presence of severe pulmonary hypertension, and especially when there is a reversal of flow through the ductus, that is, right to left, the continuous murmur will be replaced by a systolic murmur and an early diastolic murmur of pulmonary insufficiency.

In the presence of shunt reversal, the patient often exhibits cyanosis of the toes but not of the fingers. In such cases, simultaneous arterial blood samples from the right brachial and a femoral artery should be taken and will enable the diagnosis to be made. The sample from the femoral artery will be considerably lower in oxygen content than the sample from the right brachial artery. The patient with shunt reversal, which occurs because of an extremely high pulmonary vascular resistance, does not tolerate operative repair of the ductus. When atypical cases are referred for further investigation, it should be realized that right heart catheterization will demonstrate the left-to-right aortic pulmonary shunt if it exists, but, unless the ductus itself is catheterized, the exact nature of the lesion is not certain. In these cases, a retrograde aortogram with the injection of contrast medium into the root of the aorta will demonstrate the type of lesion which is present.

Ventricular septal defect. This is another relatively common isolated congenital defect. Its repair by the use of open-heart techniques with the heart-lung machine is now attended with much success, and, in the younger patients, in the absence of severe pulmonary hypertension accompanied by bidirectional shunting, the operative mortality should not exceed 5 per cent.

The lesion may be roughly classified as mild (*maladie de Roger*), moderate, severe, and pulmonary hypertensive. As in atrial septal defects,

the lesion and its physiologic effects may be assessed with considerable accuracy in the office.

The mild case is unassociated with symptoms and has a small left-to-right shunt. The physical signs are a normal pulse, a normal cardiac apex beat, normal heart sounds, and a pansystolic murmur and accompanying thrill with maximum intensity at the third and fourth left intercostal spaces close to the left sternal edge. The thrill is rarely absent, and its absence should render the diagnosis doubtful.

In moderate and severe cases, there may be underdevelopment, fatigue, and breathlessness, but symptoms may be absent. The pulse may be small. The apex beat is left ventricular in type and hyperdynamic, and, in severe cases, a right ventricular lift is felt to the left of the sternum in the third and fourth spaces. The heart sounds may be normal, and normal splitting of the second heart sound is heard. Wide splitting of the second heart sound, as in atrial septal defect, is notably absent. With a large left-to-right shunt, a loud third heart sound may be heard at the apex. The pansystolic murmur and thrill are present and, in addition, a mitral diastolic murmur due to a large, rapid flow through the mitral valve can be heard in severe cases. This sign is characteristic of a large shunt and is com-

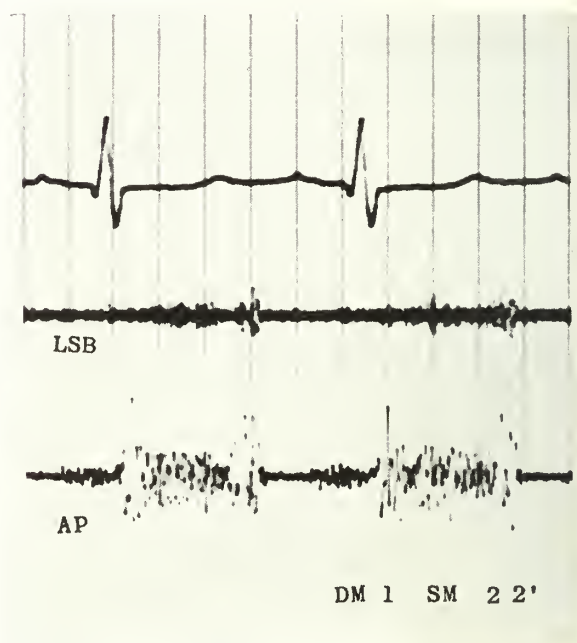


Fig. 1. Phonocardiogram from a severe case of ventricular septal defect showing at the apex (AP) the loud systolic murmur (SM) and the mitral diastolic filling murmur (DM).

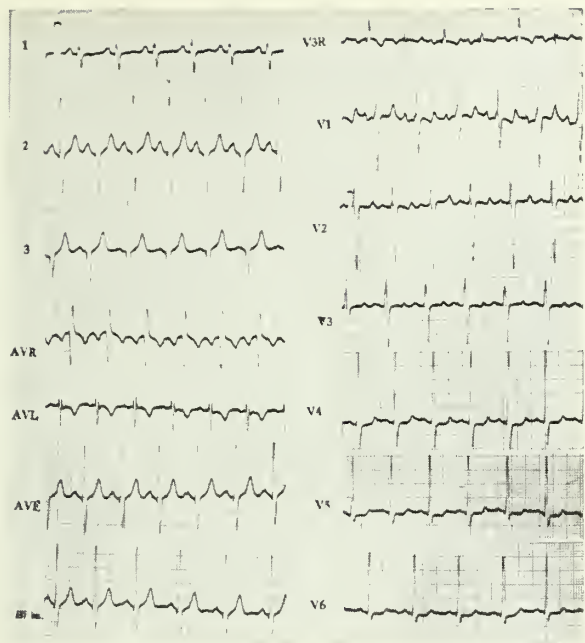


Fig. 2. Electrocardiogram from a patient with ventricular septal defect showing right axis deviation and evidence of right ventricular enlargement (tall R V₁) and left ventricular enlargement, QR, and low T in V₆.

parable with the tricuspid diastolic murmur of atrial septal defect (figure 1).

In the pulmonary hypertensive group, physiologic studies will demonstrate a pulmonary artery systolic pressure equal to the systemic systolic pressure; some patients may maintain a left-to-right shunt, while others exhibit a still greater pulmonary vascular resistance and manifest a bidirectional or even a total right-to-left shunt with cyanosis. In these patients, a marked right ventricular lift is present. The second heart sound is loud in the pulmonary area, and a pulmonary diastolic murmur due to pulmonary valvular incompetence may be present. The systolic thrill and murmur will be less pronounced than in the moderate or severe group and, in fact, are likely to be absent in the cyanotic patients.

The electrocardiogram in ventricular septal defect tends to follow the physiologic effects of the defect. It is normal in the mild cases and shows evidence of both right and left ventricular enlargement in moderate and severe cases. The Q and R waves in the left precordial leads are much greater than those seen in patients with isolated right ventricular hypertrophy (figure 2).

Radiologically, the appearances are those of an intracardiac left-to-right shunt. These appear-

ances may be characteristic, though not necessarily diagnostic. The features of a severe defect are a relatively small aortic knob, a large pulmonary artery and main branches, pulmonary plethora, and enlargement and vigorous activity of both ventricles. In the milder examples, these changes are present but less marked (figure 3).

The mildest cases, comprising a relatively small group, probably do not require surgical repair, while those patients with shunt reversal are beyond help at the present time. The majority of patients, however, should be considered for open-heart repair.

At present, we advise that right heart catheterization should be done in all cases to confirm the diagnosis and to assess the severity of the shunt and its effects in comparison with the clinical findings. This procedure not only helps in confirmation of the diagnosis but is an aid to the differential diagnosis which arises in the milder cases of ventricular septal defect. Cardiac catheterization enables the distinction to be made between lesions such as mild pulmonary stenosis, mild aortic or subaortic stenosis, mitral insufficiency, mild atrial septal defect, and a combination of ventricular septal defect and pulmonary stenosis without cyanosis. Some patients diagnosed as having a ventricular septal defect have no recognizable defect when physiologic studies are made, and the true cause of their cardiac murmur often cannot be stated with certainty.

Atrial septal defect. Flap-like or probe patency of the foramen ovale is common and does not constitute a cardiac lesion, since it does not permit intracardiac left-to-right shunting. Atrial septal defects imply defects in the septum, usually at least $\frac{1}{2}$ cm. in diameter and commonly much larger, through which shunting of blood from the left to the right atrium occurs. The common defect is situated in the region of the fossa ovalis but may be found in other parts of the septum. Low defects, the lower margins of which are formed by the upper edge of the ventricular septum, are known as septum primum defects and are usually complicated by clefts in the mitral valve permitting mitral regurgitation. More complex lesions involve the addition of a ventricular septal defect resulting in a complex lesion known as a partial common atrial ventricular canal defect.

Atrial septal defects, the effects of which are sufficient to be clinically recognizable, should be closed surgically. The left-to-right shunt is usually fairly large, and dilatation of the right heart occurs. In later life, usually by about the fourth decade, pulmonary hypertension due to



Fig. 3. Roentgenogram of severe ventricular septal defect showing cardiac enlargement, enlargement of the pulmonary artery segment, a relatively small aortic knob, and marked pulmonary plethora.

pulmonary vascular sclerosis and thrombosis is common.

The clinical picture of an atrial septal defect is clear and diagnosis is usually easy. The major findings include the following:

1. The arterial pulse is usually normal or small in volume.
2. A left parasternal systolic lift of right ventricular enlargement is usually found.
3. The second heart sound in the pulmonary area is markedly split due to separation of the aortic and pulmonary elements beyond the range of normal. This is due to prolongation of right ventricular systole resulting from its increased diastolic volume. The split does not change during the respiratory cycle. This "fixed split" is a cardinal auscultatory sign of atrial septal defect.
4. A systolic murmur at the pulmonary area due to the large volume of flow ejected with each systole is usually to be heard, but it is uncommonly accompanied by a thrill.
5. A short diastolic murmur is present at the lower end of the sternum. This murmur is due to the rapid inflow of a large volume of blood from the right atrium to the right ventricle. The presence of such a murmur implies a large left-

to-right shunt. This murmur may sometimes be accentuated or brought out on deep inspiration or as a result of exercise.

In the presence of marked pulmonary hypertension, accentuation of the pulmonary element of the second sound may be pronounced, and a high-pitched early blowing diastolic murmur of pulmonary valvular incompetency may occur.

An ostium primum defect may be suspected when a separate loud pansystolic murmur of mitral valvular insufficiency is present in addition to the aforementioned physical signs. In some ostium primum cases, the apex beat becomes circumscribed and thrusting, indicating left ventricular enlargement.

The typical electrocardiogram of an atrial septal defect will show the pattern of incomplete right bundle-branch block, thought to indicate right ventricular enlargement, and dilation (diastolic overload). In cases with marked pulmonary hypertension, more florid evidence of right ventricular hypertrophy occurs. Ostium primum and partial atrial ventricular canal defects may be suspected when the same pattern is complicated by left axis deviation and, sometimes, by evidence of left ventricular enlargement (figure 4).

Radiologically, the lesion is characterized by a small or normal aortic knob, an enlarged pulmonary artery with large main branches, and enlargement of the right ventricle and right atrium.

It is our current practice to advise right heart catheterization in all cases. This is done because of the relatively high incidence of anomalous pulmonary venous drainage into the right atrium or superior vena cava in association with the atrial defect. The procedure frequently enables the complication to be diagnosed. In pulmonary hypertensive cases, the diagnosis may be more uncertain, and right heart catheterization is again a useful procedure.

Anomalous pulmonary venous drainage into the right atrium or superior or inferior vena cava. Atrial septal defects may be complicated by the drainage of one or more pulmonary veins into the right atrium or superior vena cava. The diagnosis is sometimes established at the time of cardiac catheterization, when the catheter enters the anomalous vein or veins. A selective angiogram with injection into the right ventricle or pulmonary artery may demonstrate the anomalous connections of the pulmonary veins during the late phase of the series.

Partial anomalous pulmonary venous drainage may exist as an isolated anomaly and will manifest a clinical picture like that of an atrial septal

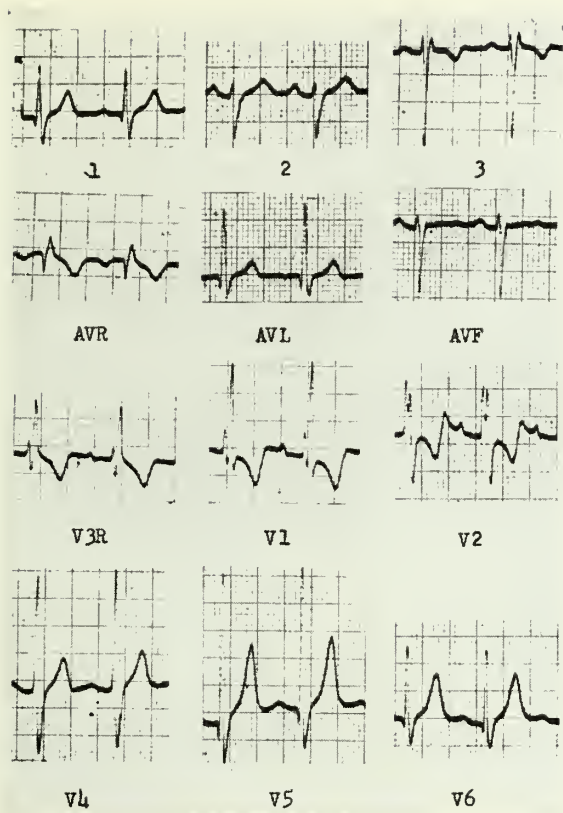


Fig. 4. Electrocardiogram of an ostium primum type of atrial septal defect complicated by mitral insufficiency. Note the typical rSr' pattern in V₃R and V₁ seen in atrial septal defects but complicated by left axis deviation and a tall QR in V₁, indicating concomitant left ventricular enlargement.

defect, and diagnosis of the isolated lesion from the combination of the two is most difficult, even with the aid of special technics.

Total anomalous pulmonary venous drainage may occur as a complication of other cardiac defects or as an isolated anomaly in association with an interatrial communication. The diagnosis of this lesion is usually dependent upon right heart catheterization and angiocardiography. This anomaly is quite rare in comparison with other conditions causing left-to-right shunts, and office diagnosis is not often possible. Most cases are usually seen during infancy, and relatively few patients with this lesion survive to adult life.

Pulmonary stenosis. Uncomplicated isolated pulmonary stenosis is one of the more common congenital heart lesions, and it ranges in degree from mild to extremely severe. The stenosis is valvular in the great majority of patients, but stenosis of the infundibulum of the right ventricle is sufficiently frequent to be considered at all times in differential diagnosis. Cyanosis is

absent except in the presence of a severe stenosis with a very high right ventricular pressure leading to a high atrial pressure and the forcing open of a patent foramen ovale and, consequently, to a right-to-left interatrial shunt. Symptoms may be absent even in severe stenosis and are not used as a primary guide to the need for surgery.

The diagnosis is usually simple and is based on evidence of right ventricular enlargement with a systolic murmur and thrill maximal at the pulmonary area. The second sound is soft at the pulmonary area because the murmur may obscure the aortic element of the second sound, while the pulmonary element is diminished. Nevertheless, the second heart sound should be most carefully auscultated, since a soft, delayed pulmonary element due to prolonged right ventricular systole and low pulmonary diastolic pressure is a feature of pulmonary stenosis. The delay in the pulmonary element is greatest with the more severe degrees of stenosis.

Phonocardiography, which has been of such great service in elucidating the significance of murmurs and sounds, is of value in analyzing the second heart sound in pulmonary stenosis.

The electrocardiogram reveals varying degrees of right ventricular enlargement, severe cases showing the most striking changes of all congenital lesions.

The roentgenogram usually shows varying degrees of right ventricular enlargement and enlargement of the main pulmonary artery without plethora of the lung fields. In severe cases, the lungs show obviously diminished vascularity.

Since surgical intervention is indicated in the moderately and extremely severe cases, right heart catheterization is usually indicated. This not only gives a measure of right ventricular and pulmonary arterial pressures but may indicate the presence of infundibular stenosis, and it seeks out the cases associated with septal defects. Conservatively, it is suggested that patients with right ventricular systolic pressures of 80 to 90 mm. Hg (about four times normal) or more should be operated on, preferably by open-heart technics, which permit visual exposure of the pulmonary valve and enable the infundibular stenosis to be dealt with if necessary.

Angiocardiography performed by the selective technic, in which a thin-walled cardiac catheter is placed in the lower part of the outflow tract of the right ventricle and contrast medium injected under pressure, is often of great assistance in demonstrating the presence of valvular and infundibular stenosis and is helpful in differentiating the diagnosis from the tetralogy of Fallot in cyanotic patients (figure 5).

Tetralogy of Fallot. This is the most common of the cyanotic congenital heart conditions seen in childhood and adult life. The condition comprises a large ventricular septal defect, an overriding of the defect by the aorta, infundibular and, sometimes, valvular pulmonary stenosis, and right ventricular hypertrophy.

Cyanosis is present during the first year of life, but, in a few cases, the cyanosis is minimal or even absent. Clubbing of the fingers and toes is present in cyanotic children and adults. Dyspnea on effort is common, and a history of squatting is frequent and of some diagnostic value. Physical examination usually reveals the cyanosis and clubbed fingers. The apex beat of the heart is usually in the normal position. A thrill is present along the left sternal border in many cases, and a relatively short but usually harsh systolic murmur can be heard over this region and at the pulmonary area. The second heart sound is audible at the apex, over the left sternal border, and usually at the aortic and pulmonary areas. It is often quite loud and almost always pure and unsplit. It is due entirely to aortic valve closure. Unlike pure pulmonary valvular stenosis, splitting of the second heart sound is rarely audible. The relatively short systolic murmur does not obscure the aortic second sound as may the longer murmur in severe pure stenosis. These two auscultatory signs may be used as a differential diagnostic aid.

The electrocardiogram shows right axis deviation and unequivocal evidence of right ventricular enlargement. In general, the changes are less marked than those seen in severe pure pulmonary stenosis, and, in cyanotic cases, this may be a clue to the differentiation.

The x-ray picture in children and adults usu-

ally shows minimal or no cardiac enlargement with a "tipped-up apex" and a hollow pulmonary artery segment. In cases with valvular stenosis, a poststenotic dilation of the pulmonary artery may be visible. The aortic knob is usually normal or prominent, since it carries a large output of blood from both the left and right ventricles. In about 25 per cent of cases, the aortic arch is right-sided. The lung fields may appear relatively normal or may show a diminished vascularity.

The diagnosis can usually be made clinically, but cardiac catheterization and angiocardiology help to exclude cases with pulmonary artery atresia or unilateral absence of the pulmonary artery. Angiocardiology will demonstrate the occasional case of transposition of the great vessels, diagnosed as tetralogy, and will enable the anatomy of the right ventricular outflow tract and the pulmonary valve area to be visualized.

The operation of choice for this condition is open repair of the ventricular septal defect and relief of the stenosis of the infundibulum and pulmonary valve, using total cardiopulmonary bypass. The Blalock-Taussig or Pott's operations will probably be reserved in the future for the temporary treatment of severely disabled infants who are not yet large enough for complete and open repair of the defect.

Coarctation of the aorta. Patients with coarctation of the aorta are most commonly seen because of a cardiac murmur in the case of children and hypertension in the case of older patients. The preductal coarctation, in which the lower half of the body is supplied with blood by the right ventricle, is uncommon and only rarely seen beyond infancy. The common postductal type, however, is not a rare lesion and is seen more often in males than in females. The condition is characterized by an elevated systolic blood pressure in the upper extremities and a lower blood pressure, as measured by the cuff, in the legs. The upper part of the body is often well developed in comparison with rather slender legs. Vigorous pulsation of the right carotid artery is often a clue to the diagnosis in young children. Collateral vessels in the interscapular region and along the side of the chest are occasionally seen in children but not as frequently as in adults. The uncomplicated coarctation may show no cardiac enlargement, but the left ventricular apex beat usually indicates hypertrophy of that chamber. The heart sounds are noticeable in that the second sound is accentuated at the aortic area, and there is often a so-called ejection sound early in systole. A rather delayed systolic murmur over the mid-precordium is usually audible. It is usually not

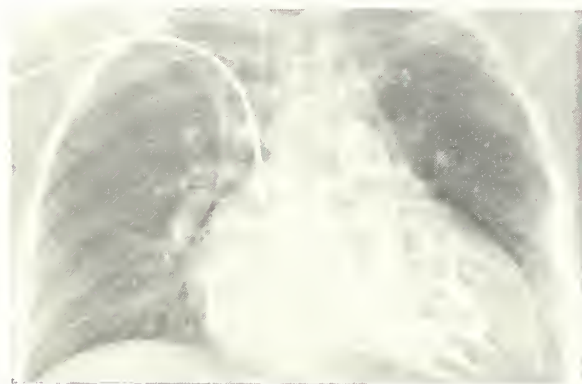


Fig. 5. Selective angiogram in patient with isolated pulmonary valve stenosis. Contrast medium can be seen in the pulmonary artery, and the dome-shaped stenotic valve may be clearly made out as a crescent-shaped translucent line.

loud enough to produce a thrill. A very loud murmur should raise the suspicion of some complicating lesion.

The electrocardiogram is often normal or may show signs of left ventricular enlargement. However, particularly in young children, the pattern of so-called incomplete right bundle-branch block or of minimal right ventricular enlargement is not uncommon even in uncomplicated coarctation of the aorta.

Radiologic examination may show some enlargement of the left ventricle accompanied by smallness of the aortic knob and possibly with the shadow of the dilated left subclavian artery above it. Rib notching should be sought but is not usually seen before 6 to 8 years of age. In general, it is considered today that all cases of uncomplicated coarctation should be surgically repaired. The operation is optimally performed between the ages of 8 and 15 years. In older patients, particularly in patients 25 to 30 years of age and older, vascular changes make the operation more difficult, but many successful repairs have been done in these and in even older age groups. In an uncomplicated case, special investigations are not indicated. However, the suspicion of a complication would suggest the need for further investigation. Coarctation of the aorta may be complicated by a bicuspid aortic valve which may be stenotic. Aortic insufficiency, ventricular septal defects, and patency of the ductus arteriosus are other associations, and right heart catheterization may be indicated. In addition to the presence of murmurs which might indicate a complicating lesion, signs of severe right ventricular hypertrophy on the electrocardiogram or of severe left ventricular hypertrophy with marked T wave changes are also indications for further investigation.

Aortic stenosis. Congenital aortic stenosis may be either valvular or, less commonly, subvalvular in the form of a subvalvular fibrous ring. Pure congenital aortic stenosis is not one of the more common conditions, but it is far from rare. Aortic stenosis in almost all young children and in many older children and adults is congenital. Commonly, the condition is well tolerated for many years, but, even in young children, considerable cardiac enlargement, electrocardiographic evidence of left ventricular hypertrophy, and serious symptoms may occur. The presence of symptoms indicating left ventricular failure is always serious in aortic stenosis and serves as an indication that relief is necessary. If good results are to be obtained from cardiac surgery for aortic stenosis, patients will probably have

to be operated on before left heart failure begins. The clinical diagnosis is usually made from the presence of left ventricular hypertrophy and a systolic murmur and thrill at the aortic area. Only in the more severe cases are the classical anacrotic pulse, diminished aortic second sound, and narrowed pulse pressure found. The electrocardiogram usually shows evidence of left ventricular hypertrophy, depending upon the effect of the lesion upon the left ventricle, and, similarly, the radiologic examination shows varying degrees of left ventricular enlargement. A markedly hypertrophic left ventricle may be present in aortic stenosis without much enlargement of the cardiac silhouette because of the absence of any pronounced degree of ventricular dilation. The severity of aortic stenosis may perhaps best be assessed on physiologic grounds. This can be achieved by left heart catheterization, which is probably indicated in patients in whom surgery is contemplated. This may be done with a low morbidity even in children. The gradient across the aortic valve may be measured by the simultaneous recording of the femoral and the left ventricular pressure pulse. The left ventricle may be readily entered by a needle inserted near the apex through the anterior chest wall. Gradients of 40 mm. Hg and over across the valve are usually associated with a signifi-

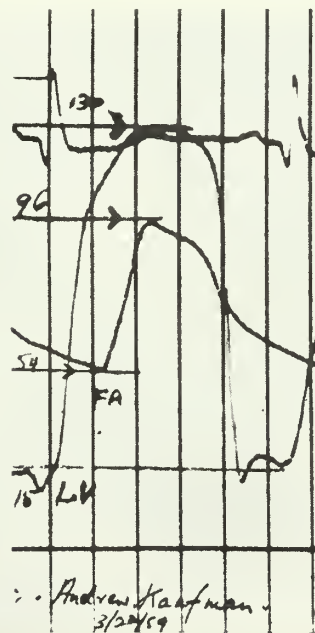


Fig. 6. Pressure pulses from a 3-year-old child with aortic stenosis. A gradient of 34 mm. Hg across the aortic valve is demonstrated. The left ventricular diastolic pressure is elevated. The patient was successfully operated on.

cant or severe degree of aortic stenosis, and such a gradient would be an indication for surgical repair (figure 6). At the present time, congenital aortic stenosis is best treated by open-heart operation using the heart-lung machine.

SUMMARY

Some of the congenital cardiac lesions amenable

to cure or alleviation by surgery have been listed and have been discussed from the point of view of the important physical signs and electrocardiographic and radiologic manifestations. With these simple measures, the physician can usually select patients who might be candidates for surgery. The ancillary investigations commonly necessary are indicated.

DIAGNOSIS of heart disease should not be based solely on an abnormal electrocardiogram, because tracings that deviate from the assumed normal may not mean that the myocardium is also abnormal. If the diagnosis is made without additional evidence, a healthy person may be converted to a cardiac invalid.

In particular, the S-T segment and the T-wave portion are poorly understood. Extracardiac causes for changes in these components of the electrocardiogram include (1) drugs, (2) electrolyte imbalance, (3) drinking of ice water, (4) emotional disturbances, (5) postprandial changes probably due to potassium shifts, and (6) hyperventilation.

S-T segment and T-wave abnormalities occur without known pathologic, physiologic, or biochemical explanation and may persist for years. If no extracardiac cause can be found and no other signs of heart disease are evident, the abnormalities must be considered to be normal variations.

A common variant is S-T segment elevations up to 4 mm. in middle to left precordial leads, simulating pericarditis. Exercise can temporarily abolish the elevation. Negative T waves in one or more of the precordial leads V_3 to V_6 may also occur in healthy subjects.

Electrocardiographic changes produced by hyperventilation are abolished by vagal blocking drugs.

Variants were noted on electrocardiograms of 5 patients without any other manifestations of heart disease. The patterns remained unchanged over one- to three-year periods. Thoracotomy and pericardial biopsy were performed for 1 patient and 2 were made cardiac invalids solely on the basis of the electrocardiogram.

In 4 of these patients, S-T segment elevation was associated with T-wave inversion. These variants were apparent under basal conditions, as well as after eating, exercise, and ingestion of Pro-Banthine.

The fifth patient had a normal electrocardiogram under basal conditions, but eating, exercise, and hyperventilation elicited T-wave inversion. The change could be prevented with Pro-Banthine.

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Surgical Management of Benign Esophageal Obstruction

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KNOWLEDGE of esophageal function, both normal and abnormal, has lagged behind advances in surgical techniques which now allow surgeons to operate freely on the esophagus. Perhaps this lack of knowledge was of minor concern when malignancy was the chief indication for esophageal surgery. Surgical extirpation to relieve dysphagia is of primary concern in such cases. Now that increasing numbers of patients are being referred to the surgeon for treatment of benign esophageal obstruction, it is more important than ever for the fundamentals of esophageal function to be understood. They should be interfered with as little as possible by the surgeon. Long survival is the rule following operation for benign esophageal disease, and an ill-conceived surgical procedure may lead to greater discomfort and disability than the patient originally experienced.

In general, the function of the esophagus is to convey ingested material from the pharynx to the stomach and to prevent the regurgitation of stomach contents into the esophagus. Safeguards exist at either end of the esophagus to implement this function. An upper esophageal sphincter guards the esophageal introitus, and an inferior esophageal sphincter guards its lowermost end.¹ The body of the esophagus is not an immobile tube but exhibits a coordinated peristaltic wave in response to deglutition, which propels ingested food throughout its length and into the stomach after relaxation of the inferior esophageal sphincter. Insofar as possible, operative procedures on the esophagus should be designed to maintain esophageal function intact.

With these basic considerations in mind, a few of the common causes of benign esophageal obstruction will be briefly reviewed. The causes are numerous, but this discussion will be concerned with achalasia of the esophagus, short esophagus with stricture, and benign tumors of the esophagus.

ACHALASIA OF THE ESOPHAGUS

Achalasia of the esophagus, more commonly and incorrectly called cardiospasm, is a disease of unknown etiology characterized by abnormal motility of the esophagus. Motor failure of the esophagus is accompanied by failure of the lower esophageal sphincter to relax on swallowing. No effective wave of peristalsis occurs in the body of the esophagus following deglutition. Pressures at the inferior esophageal sphincter are rarely increased, however, so the term "cardiospasm" is a misnomer. Dysphagia and regurgitation are the most frequent symptoms of this disease, and the distal esophageal obstruction may lead eventually to marked dilation, tortuosity, and elongation of the esophagus.

It is important to differentiate this condition from a disturbance of motility of the esophagus known as diffuse spasm, in which huge, simultaneous, repetitive contractions are present. Pain is a common feature of the latter condition and dilation of the esophagus is rare, whereas muscular hypertrophy is common.

Forceful dilation of the distal part of the esophagus and the cardia has been employed in the treatment of achalasia for many years with varying degrees of success. Results obtained at the Mayo Clinic with hydrostatic dilation indicate that permanent symptomatic relief has occurred in approximately 60 per cent of patients after a single course of therapy.² Of the remaining 40 per cent, about half experienced permanent relief after further courses, while the other half did not respond to this treatment or required periodic dilation.

Although surgical treatment of this condition was carried out as long ago as the late 1800's, only in recent years has it achieved popularity in the United States. Fortunately, the wave of enthusiasm for surgical procedures that destroy or bypass the esophagogastric junction has all but subsided. These procedures, including the Wendel cardioplasty, the Heyrovsky-Gröndahl cardioplasty, and various types of limited esophagogastric resection, have led almost uniformly to the

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development of severe and sometimes fatal esophagitis.^{3,4} Their further use in the light of present knowledge is to be condemned.

In 1913, Heller⁵ described an operation, modifications of which have been used by all who have recently reported good results in the treatment of cardiospasm. As originally described, an abdominal approach was used and 2 longitudinal myotomies were done in the distal part of the esophagus and the cardia, 1 anteriorly and 1 posteriorly. The modification of using only 1 anteriorly placed incision was proposed by Zaaijer⁶ in 1923.

Before the operative procedure is set forth in detail, it is important that the objectives of the operation be defined. In esophageal achalasia, a pressure zone exists in the lower part of the esophagus that fails to relax after deglutition. In addition, normal peristaltic activity of the body of the esophagus is absent.⁷ Surgical treatment can relieve only the obstruction resulting from failure of the lower esophageal sphincter to relax. At the present time, no known procedure can restore normal motility to the body of the esophagus. In relieving the distal esophageal obstruction, part or all of the esophago-gastric mechanism that prevents reflux must be preserved, while part or all of this mechanism that governs the passage of material from the esophagus to the stomach must be eliminated.

My colleagues and I have used a modified Heller procedure and have performed a longitudinal esophagomyotomy transthoracically in 107 patients without a death.⁸ Certain technical features of the operative procedure deserve emphasis. The incision is long, 6 to 10 cm. in length, and is confined for the most part to the distal esophagus (figure 1). The incision is extended distally onto the stomach only far enough to insure complete division of all the distal esophageal musculature. The esophageal hiatus is not incised. The vagi are undisturbed, and care is taken to narrow the esophageal hiatus behind the esophagus when indicated to prevent postoperative herniation of the stomach. If these precautions are taken, no ancillary procedures, such as pyloroplasty, are necessary, and reflux esophagitis rarely occurs.

The clinical results in the first 45 patients operated on have been presented previously, the follow-up periods varying from four months to seven years and seven months, averaging 25.1 months.⁹ Results were good to excellent in 85 per cent of patients, poor in 4 per cent, and the remaining 11 per cent were improved but still troubled by some dysphagia.

Röntgenographic examination demonstrated

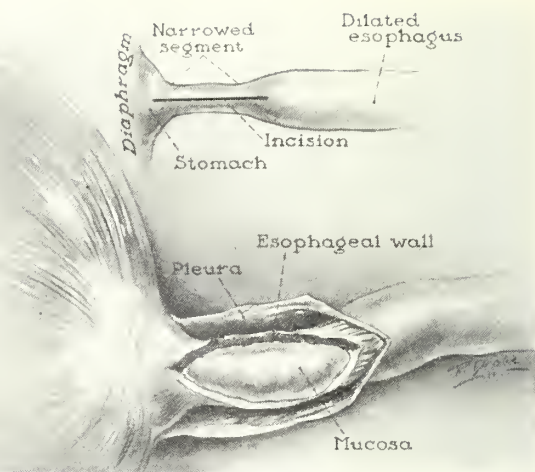


Fig. 1. Esophagomyotomy (modified Heller procedure). (Reproduced with permission from OLSEN, A. M., ELLIS, F. H., JR., and CREAMER, B.: Cardiospasm [achalasia of the cardia]. *Am. J. Surg.* 93:299, 1957.)

a recognizable decrease in esophageal caliber postoperatively in only one-half of the patients examined, and there was no correlation between clinical findings and postoperative esophageal size.

Esophageal motility studies performed at intervals after operation showed no change in motility of the body of the esophagus, but elimination of the suprahiatal portion of the esophageal sphincter was demonstrated.¹⁰

Indications for the operation are becoming more clear-cut, and it is being employed more freely at the Mayo Clinic now than in past years. There is little question that surgical treatment should be offered to those patients who fail to respond to adequate dilation or who suffer a recurrence after dilation. Patients with a huge tortuous esophagus in whom hydrostatic dilation is technically difficult and dangerous should undergo surgical intervention. When motility studies show high pressures at the sphincteric zone, muscular hypertrophy may be present and hydrostatic dilation usually fails. Patients with associated conditions requiring thoracotomy, such as diaphragmatic hernia or esophageal diverticulum, are best treated surgically. Because of the excellent results, the operation is also being employed with greater frequency than heretofore in uncomplicated cases as the primary form of treatment and may eventually become the treatment of choice for all patients suffering from this disease.

SHORT ESOPHAGUS WITH STRICTURE

Although esophageal stricture can follow a variety of precipitating events, strictures caused by

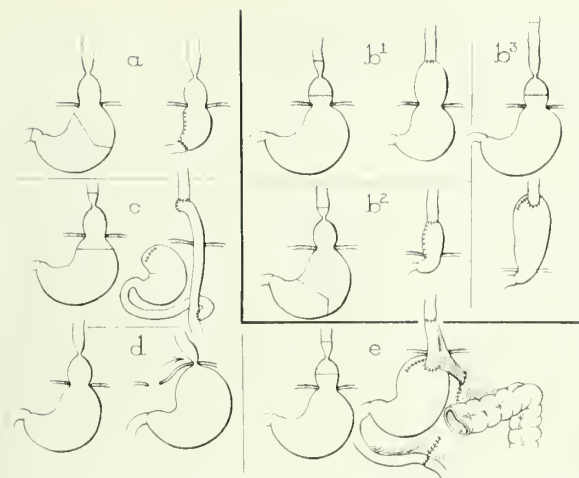


Fig. 2. Operative procedures for complications of reflux esophagitis. (a) Subtotal gastric resection. (b) Esophagogastrrectomy: 1, limited resection; 2, extensive resection of stomach; and 3, extensive resection of esophagus with high esophagogastrstomy. (c) Esophagogastrrectomy with Roux-Y esophagojejunostomy. (d) Transplantation of esophagogastric junction to dome of paralyzed diaphragm. (e) Esophagogastrrectomy with interposition of limb of jejunum between esophagus and stomach. (Reproduced with permission from ELLIS, F. H., JR., ANDERSEN, H. A., and CLAGETT, O. T.: Surgical management of complications of reflux esophagitis. *Arch. Surg.* 73:578, 1956.)

gastroesophageal reflux are most common. When the gastroesophageal mechanism is disturbed and reflux of gastric juice into the esophagus occurs, inflammation and ulceration of the terminal esophagus may develop. If severe and untreated, such lesions may progress to scarring, stricture formation, and shortening of the esophagus. The term "short esophagus" is used to describe the condition in which the esophagogastric junction lies above the diaphragm and cannot be restored to its normal subdiaphragmatic position. It is an acquired condition in all but rare instances.

Reflux esophagitis has many causes, but an esophageal hiatal hernia is the predisposing factor in more than half the cases.¹¹ Operations that disrupt the integrity of the gastroesophageal mechanism uniformly lead to esophagitis, and this is the next most common cause of reflux esophagitis. Other conditions favoring gastroesophageal reflux, such as postoperative vomiting, vomiting of pregnancy, pyloric obstruction, or idiopathic incompetency of the cardia, can result in esophagitis and esophageal stricture.

Although medical management may control the symptoms of some patients with a short esophagus and stricture, it frequently is inadequate or fails entirely. When it fails and surgical treatment must be undertaken, the surgeon must

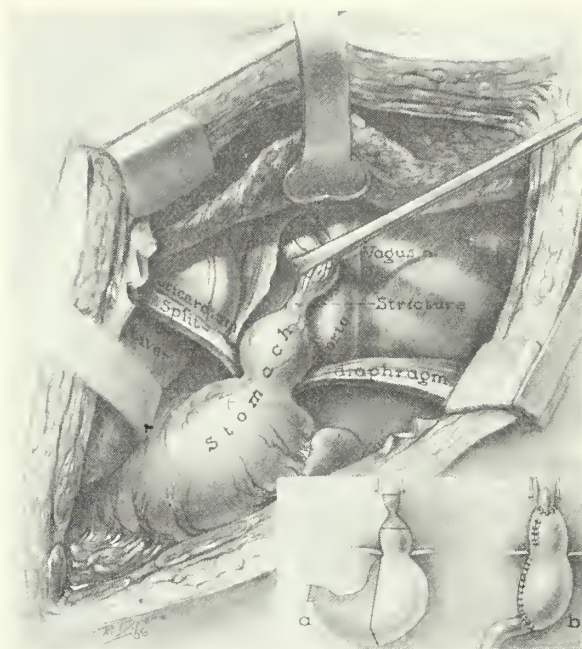


Fig. 3. Esophagogastrrectomy and antral excision for short esophagus with stricture. (a) Diagram indicating parts to be resected. (b) Diagram of completed operation. (Reproduced with permission from ELLIS, F. H., JR., ANDERSEN, H. A., and CLAGETT, O. T.: Treatment of short esophagus with stricture by esophagogastrrectomy and antral excision. *Ann. Surg.* 148:526, 1958.)

choose from a variety of operative procedures (figure 2). Early experiences at the Mayo Clinic with some of these operations, including the conventional esophagogastrrectomy, Roux-Y esophagojejunostomy, and subtotal gastric resection, were unsatisfactory. The jejunal interposition operation of Merendino and Dillard¹² is a physiologic operation which overcomes many of the disadvantages of the aforementioned procedures. I have had no experience with this particular procedure but continue to employ esophagogastrrectomy and antral excision when indicated.¹³

The latter procedure consists of resection of the strictured zone, bilateral vagotomy, and distal gastric resection with removal of the antrum. Gastrointestinal continuity is reestablished by esophagogastrstomy and gastroduodenostomy (figure 3). Following this procedure, the stricture is removed, a satisfactory gastric reservoir remains, and normal continuity of the alimentary tract is maintained. In addition, bilateral vagotomy eliminates the cephalic phase of gastric secretion, and resection of the antrum eliminates the gastric phase. Thus, gastric acidity is greatly reduced, only the intestinal phase remaining.

The operative procedure is extensive and should be employed only when clearly indicated.

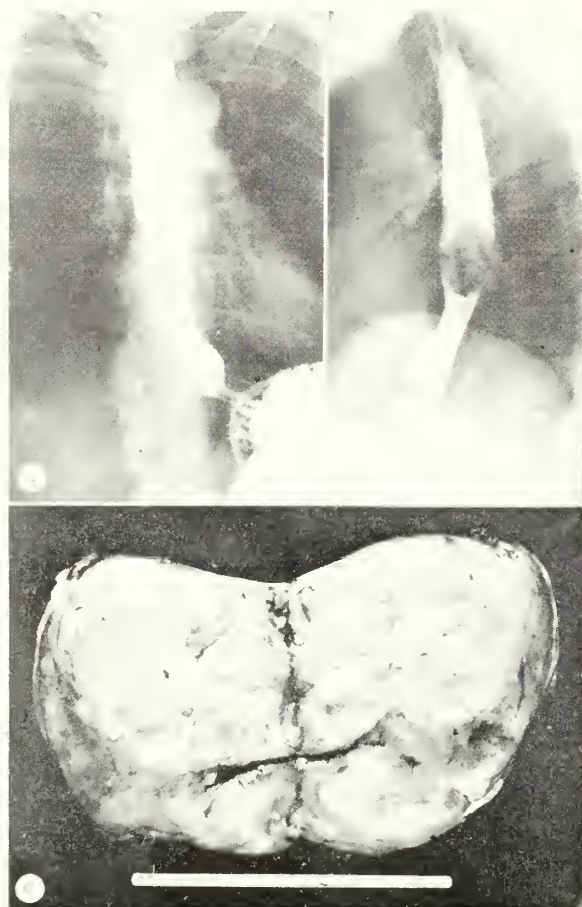


Fig. 4. Leiomiyoma of the esophagus. (a and b) Esophagograms. (c) Gross specimen.

At the present time, patients with esophageal stricture are initially treated in a conservative fashion. Efforts are made to counteract gastric acidity and to prevent gastroesophageal reflux by diminishing intraabdominal pressure by attention to diet and avoidance of tight clothing. Dilation of the strictured part is carried out as indicated. When, as so often happens, these measures fail and dysphagia recurs promptly after dilation, operative intervention is mandatory.

More than 25 patients have undergone the operative procedure, which resulted in 1 postoperative death. Postoperative complications were few and of no permanent significance. Early results in some of these patients previously reported upon indicated that all but 1 had been relieved of their preoperative symptoms.¹¹ By its very nature, the operative procedure permits gastroesophageal reflux, but all patients have been achlorhydric and there is little tendency for esophagitis to recur. Because of the absence of a gastroesophageal sphincter, patients are ad-

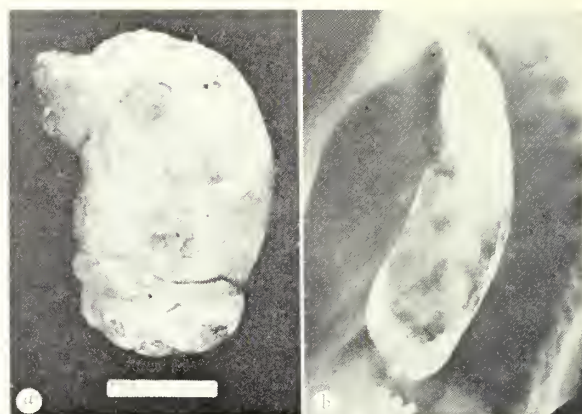


Fig. 5. Fibrolipoma of the esophagus. (a) Gross specimen. (b) Esophagograms. (Reproduced with permission from BERNATZ, P. E., SMITH, J. L., ELLIS, F. H., JR., and ANDERSEN, H. A.: Benign, pedunculated intraluminal tumors of esophagus. *J. Thoracic Surg.* 35:503, 1958.)

vised to sleep with the heads of their beds elevated to avoid regurgitation.

Early in our experience with this operative procedure, it was employed not only for patients with esophageal stricture and a shortened esophagus but also for those without stricture with severe symptoms of bleeding or pain or both. Finney pyloroplasty has been employed recently in the surgical management of the latter patients in an attempt to facilitate gastric emptying and decrease gastroesophageal reflux.

BENIGN ESOPHAGEAL TUMORS

Benign tumors of the esophagus are relatively uncommon. Malignant lesions occur at least 100 times more frequently.¹⁵ Benign tumors can be divided into 2 main categories: (1) smooth muscle extramucosal leiomyomas, and (2) pedunculated intraluminal fibrolipomas.

Smooth muscle tumors of the esophagus occur most commonly in the middle or lower third of the esophagus. A few involve the esophagogastric junction. They are submucosal in location and ulceration of the surface mucosa rarely occurs, in contrast to leiomyomas of the stomach. Johnston and associates,¹⁶ of the Mayo Clinic, reported 21 cases in 4 of which the esophagogastric junction was involved and in 4 of which the tumors were multiple.

Symptoms are not marked. Indeed, almost half of the patients seen at our institution were asymptomatic. This is in contrast to patients with leiomyosarcoma of the esophagus, who usually have a severe degree of dysphagia. Diagnosis is best accomplished by radiologic and esophagoscopy examinations. A mediastinal mass can frequently be detected on routine anteroposterior thoracic roentgenograms. With the

aid of a barium swallow, a smooth defect in the outline of the esophagus is apparent (figure 4a and b). When examined esophagoscopically, the tumor seems to protrude into the lumen of the esophagus. The overlying mucosa is intact. In order to simplify a subsequent operative procedure and to maintain mucosal integrity, the esophagoscopist should refrain from biopsy of the lesion.

Leiomyomas of the esophagus may be round, oval, or U-shaped, or they may completely encircle the esophagus (figure 4c). Under the microscope, the pattern of interlacing bundles of spindle-shaped cells is typical.

The majority of leiomyomas can be treated by simple enucleation without damaging the esophageal mucosa. This was accomplished without a death in all the cases of Johnston and associates.¹⁶ When the lesion is more extensive, more particularly when it has extended to involve the stomach, a resective procedure may be indicated.

Polypoid intraluminal tumors were among the first benign esophageal tumors to be described. Although they occur much less frequently than leiomyomas of the esophagus, they are of considerable interest because of the sensational manner in which they may appear and because, if untreated, they may result in the death of the patient. In the collected review by Totten and associates,¹⁷ it is reported that one-third of the patients died as a direct or indirect result of tumor.

These tumors usually arise at a high level, either from the cervical or high thoracic esophagus. They are attached by a fairly long pedicle arising from the wall of the esophagus (figure 5a). Multiple tumors have been known to occur. The mucosa over the surface of the tumor is usually intact, although ulceration may develop. Microscopically, these tumors are composed of a mixture of loose, fibrous tissue associated with myxomatous and fatty changes. The descriptive term "fibrolipoma" seems appropriate for their designation.

Obstruction of the esophagus leads to dysphagia, and regurgitation of the tumor through the mouth may occur, much to the consternation of the patient. This occurred in 4 of the six patients reported on by Bernatz and associates.¹⁸

Thoracic roentgenograms usually reveal mediastinal widening. Esophageal roentgenograms made after ingestion of barium show dilation of the esophagus, and an incorrect diagnosis of esophageal achalasia is frequently made (figure 5b). Such a diagnosis was made in 4 of the 6 cases just mentioned. Esophagoscopy permits a more accurate diagnosis. Surgical excision is the

treatment of choice. The site of origin should be accurately determined preoperatively in order to select the proper operative approach. Only 2 of our patients required thoracotomy, the remainder of the tumors being removed transorally or transcervically, and all 6 underwent satisfactory removal without recurrence.

SUMMARY

Since patients with benign esophageal obstruction are being referred to the surgeon with increasing frequency, it is important that fundamentals of esophageal function be understood. Surgical procedures that minimize disturbances of esophageal function should be selected.

Achalasia of the esophagus, short esophagus with stricture, and benign tumors of the esophagus are among the more common benign causes of esophageal obstruction. All may be benefited by operation, and indications for surgery as well as details of surgical technic have been discussed.

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Renal Ischemia as a Cause of Hypertension

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HYPERTENSION is a common finding on physical examination. Establishment of the cause of blood pressure elevation, whenever possible, is the most valuable key to the proper therapy for each patient. That patients with renal disease often have hypertension has long been known. In 1836, Bright¹ first described the occurrence of cardiovascular changes related to hypertension in patients with chronic renal disease. It was in 1898 that Tigerstadt and Bergman found a substance in the cortical extract of rabbits' kidneys and in renal vein blood which produced profound and sustained hypertension on intravenous injection.

A number of experimental procedures that produced inconclusive results followed. Then, in 1934, Goldblatt² found that controlled hypertension could be produced by application of silver clamps to the renal artery. His discovery was followed by a number of projects that demonstrated the nature of the substance produced by the kidney, called renin, and its activation by a part of the serum alpha-globulin producing a powerful pressor agent called angiotonin.³⁻⁶ Clinical application of these principles was begun in 1937 with the demonstration of relief of hypertension following nephrectomy in certain patients with unilateral pyelonephritis. In 1956, Smith⁷ reviewed a large number of reports and found that a successful result, as determined by blood pressure of less than 140 mm. Hg systolic and 90 mm. Hg diastolic for at least one year, was obtained in 26 per cent. In 1954, Howard and associates⁸ and, in 1956, Pontasse and Dustan⁹ demonstrated that hypertension in the human being could result from renal vascular disease and could be cured by either correction of the existing defect or nephrectomy, providing the lesion involves only one kidney. Pontasse also described cases of hypertension due to renal artery ischemia in which intravenous pyelograms failed to show evidence of unilateral renal dis-

ease. Diagnosis in these cases was made preoperatively by aortography. The relative safety of aortography was established in a large series by McAfee and Willson¹⁰ and has become an accepted procedure in diagnosis of renal vascular lesions.

Before going into the pathologic aspects of a renal artery lesion, reexamination of some of the concepts of vascular abnormalities in hypertensive patients is worthwhile. Meaney and associates¹¹ pointed out that in a series of 29 patients, 16 of whom were hypertensive, arterial patterns after angiography of all of the kidneys of the hypertensive patients exhibited a characteristic abnormality, namely, diminution or lack of filling of the interlobular arteries—a finding which is strikingly similar to that observed in experimental animals with renal ischemia. This anomaly was not evident in renal angiograms of 13 normotensive patients.

Besides the hypertensive vascular disease, that is, nephrosclerosis, we are especially interested in obstructive renal artery lesions, which can be divided into two main groups: extrinsic compression and intrinsic arterial disease. Both are capable of causing renal hemodynamic changes and, possibly, of initiating the release of renal pressor substances. Extrinsic causes of compression of the renal artery include aortic aneurysm and retroperitoneal tumor. The intrinsic type of renal artery disease includes embolism, thrombosis, developmental defects with fibrous intimal proliferation, aneurysm, arteriosclerotic plaques, and syphilitic arteritis. An embolus, almost invariably from a diseased heart, may obstruct a renal artery or one of its branches, thereby causing partial or total infarction of the kidney. Total necrosis of the kidney does not cause hypertension, but partial infarction of the kidney and reduced blood supply in other areas may cause transient reversible or severe persistent hypertension. Instances of patients who died of malignant hypertension associated with renal artery lesions are reported. One patient had a shell fragment that occluded the left renal artery, causing progressive malignant hypertension to

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which the patient succumbed shortly thereafter.

Thrombosis of the renal artery or of one of its branches may produce hypertension. When only one artery is supplying blood to the kidney, thrombosis of that artery will cause total infarction without producing hypertension. If one or more patent arteries exist or if only a branch of the renal artery is thrombosed, a portion of the kidney will remain functional; part of the kidney will have reduced blood supply, and hypertension commonly ensues. The fact should be re-emphasized that formation of hard arteriosclerotic plaques in, or at the aortic origin of, the renal artery narrows the lumen and interferes with blood flow. These plaques are usually local manifestations of generalized arteriosclerosis. Any obstruction of the lumen of the renal artery with impairment of renal blood flow may cause reduction in the size of the kidney and focal tubular atrophy, but most of the renal epithelium is preserved. As a rule, the arterioles are not significantly changed. Further reduction of the blood flowing through the kidney will result in more atrophy and, possibly, zones of infarction. It has been found that on microscopic examination of kidneys made ischemic by occlusion of arteries, tubular atrophy, increase in interstitial tissue, and a benign type of nephrosclerosis are usually encountered. Often, nonischemic kidneys supplied by patent vessels exhibit necrotizing arteriolar lesions characteristic of malignant nephrosclerosis.¹² This accords with the view that renal artery obstruction protects arterioles of the ischemic kidney, whereas, under the impact of hypertension, the kidney with the normal renal artery develops malignant nephrosclerosis. Occasionally, trauma seems to be a precipitating factor in renal arterial damage. McDonald and associates¹³ report a case in which thrombosis of the renal arteries supervened on surgical trauma and resulted in fulminating hypertension.

CASE REPORT

A 38-year-old white male truck driver was first examined on November 5, 1958, because of frontal headache of one month's duration present on awakening in the morning and gradually subsiding after the patient was up for one to two hours. Headache was accompanied by blurring of vision and fatigue. The patient had no previous history of recurrent headaches. The patient stated that he had been well previously except for an automobile accident in 1950, in which he sustained a fracture of the second lumbar vertebra without serious sequelae, and spontaneous pneumothorax following unusual exertion in 1956. Examination on both occasions, as well as one for an insurance company in 1957, had demonstrated normal blood pressure.



Fig. 1. Aortogram showing distortion and stenosis of right renal artery

Physical examination revealed a well-developed, well-nourished white male. Temperature was 98.6° F., pulse 88, and blood pressure 240/135 in the right arm, 235/130 in the left arm, and 250/140 in the left leg. Fundi demonstrated grade III hypertensive changes, with scattered soft exudates and flame-shaped hemorrhages bilaterally. The rest of the physical findings were normal.

Hemoglobin was 15.3 gm., and the leukocyte count was 9,900, with 64 segmented forms, 32 lymphocytes, and 3 band forms. The urine had a specific gravity of 1.018, an acid reaction, and no albumin or sugar; microscopic examination showed an occasional white blood cell. Serum sodium was 142 mEq., serum potassium was 3.8 mEq., and carbon-dioxide combining power was 24.2 mEq. Blood urea nitrogen was 13.7 mg. per cent. Urinary catecholamines were reported normal. Blood pressure did not fall after intravenous injection of Regitine. The electrocardiogram was interpreted as showing slight left ventricular hypertrophy. The posteroanterior chest roentgenogram was interpreted as normal. An intravenous urogram on November 10, 1958, was interpreted as showing a slight delay in the appearance of the dye on the right side in the five-minute x-ray film but otherwise was normal. On November 11, 1958, an aortogram was done under general anesthesia, during which time blood pressure fell to its lowest preoperative level, 160/110. The aortogram demonstrated distortion and stenosis of the right renal artery close to its origin at the aorta and poststenotic dilation (figure 1).

Operation was performed on November 20, 1958. Through a right subcostal incision, a transperitoneal approach was utilized. Right and left kidneys were normal by palpation. The duodenum and head of the pancreas were mobilized. After both renal veins were visualized, the right renal vein and vena cava were elevated to expose the right renal artery. Although this artery ap-

peared normal on inspection, palpation revealed a marked thrill originating 1.5 cm. from the aorta and diminished pulsation distal to the stenosis. To further substantiate diagnosis, arterial pressure readings were done. A 20-gauge needle attached to a 60-cm. spinal manometer system was introduced into the right renal artery distal to the stenosis. When the manometer system was opened, blood barely dribbled over the manometer at 60 cm. Next, the needle was introduced into the proximal artery between the aorta and the stenosis. When the manometer system was opened, blood shot out the top of the manometer almost to the overhead operating table light and so could not actually be measured. A decision was made to do a right nephrectomy, which was accomplished by individual ligation of the vessels and ureter and removal of the kidney.

During surgery, blood pressure varied from 180/110 to 160/110 and at the time of closure was 210/120. Within fifteen minutes after completion of surgery, blood pressure fell to 80/40. At this time, the patient was given subcutaneous Aramine and required 0.5 cc. of Aramine every half hour to maintain blood pressure at 105/80. Accordingly, five hours after surgery, an intravenous infusion containing 4 cc. of norepinephrine per 1,000 cc. was given. The patient demonstrated extreme lability of blood pressure on this program, with values ranging from 120/90 to 280/140. Because preventing wide fluctuations in his blood pressure with small variations in the rate of the intravenous infusion was very difficult, intravenous Aramine was reinstituted and norepinephrine was discontinued. Blood pressure was then maintained at 120/90, with gradually decreasing amounts of intravenous Aramine; it was possible to discontinue Aramine three days after surgery, at which time the patient's blood pressure remained stable at 130/90. The postoperative course was otherwise uneventful, and blood pressure was 135/90 on December 9, 1958, and was 140/90 on January 9, 1959. Funduscopic examination on the latter date showed remarkable clearing of the retinal hemorrhages and very few exudates. Blood pressure on December 12, 1959, was 140/90.

On gross pathology, the kidney weighed 151 gm. Its capsule stripped fairly easily, and the surface was smooth and grayish red. The renal artery stemmed from the usual anatomic location. The vessel was 3 cm. long. A suture was placed close to its "aortic" end, and a thick, yellowish gray, somewhat glistening, conspicuous narrowing was observed (figure 2). A few millimeters away, toward the kidney, another section showed grossly complete obliteration of the vessel which extended within the artery for about 3 mm. From then on, the vessel remained patent and was somewhat dilated distal to the obliterated area. On sectioning, there was also a stump of a meter, 2 cm. long and about 0.3 cm. in diameter, which showed no gross abnormalities. Furthermore, no dilation of the calyces or pelvis and no salient disturbance in the renal markings existed. On microscopic analysis, sections showed the renal artery. The lumen of the arterial segment toward the aortic origin was conspicuously narrowed and had, in some sections, only a thin slitlike opening that was lined with flattened regular endothelium. No evidence of true atheromatosis was found and, in the most narrowed portion, search for fragmentation of elastic laminae was fruitless. Mural components showed much fibrosis, and, distally, the artery showed a definitely thickened, fibrous wall, with reduplication of the elastic layer in some areas. Careful microscopic analysis of random sections from the kidney exhibited no significant glomerular or tubular lesions.

Glomeruli certainly exhibited no hemorrhages in the feeding arterioles, and second- and third-grade renal arteries presented definite diffuse hyperplastic sclerosis.

DISCUSSION

Differential diagnosis of hypertension has been expanded over the years until the diseases that are known to produce high blood pressure run the gamut from mechanical causes, such as coarctation of the aorta; through endocrine disease, such as hyperthyroidism, Cushing's syndrome, and pheochromocytoma; to degenerative disease with arteriosclerotic changes in the renal vessels.

The case presented here demonstrates the value of including aortography for selected patients as part of the diagnostic survey for hypertension. An important feature of this case is the normal intravenous pyelogram. In patients with sudden onset of hypertension, rapidly progressing or malignant hypertension, or fixed high diastolic pressures, aortography can be very valuable. We attempt to restrict its use to patients who can tolerate surgical intervention should a unilateral lesion be demonstrated. This procedure can also be very helpful in localizing and differentiating

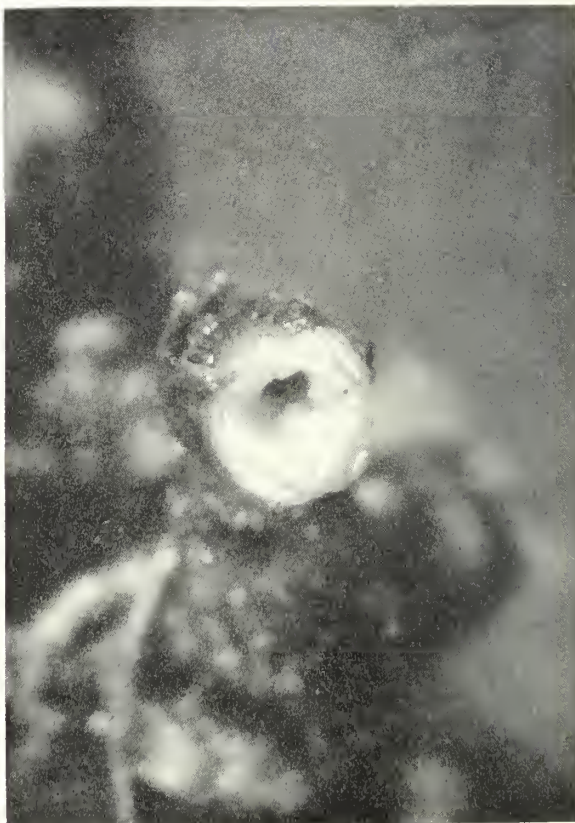


Fig. 2. Narrowing of right renal artery just proximal to the point of maximal obstruction

arterial lesions in patients with intravenous pyelograms suggesting unilateral disease. Other procedures that are of value in diagnosing unilateral renal vascular disease include differential sodium and water excretion, as described by Connor and associates,¹¹ and simultaneous counting over both kidneys following radioactive Diodrast administration, as described by Winters.¹⁵

Elucidation of the relationship of the diseased to the normal kidney is essential for proper surgical management. With the narrowing of the renal artery to the kidney and the production of hypertension, the other kidney, here called the normal kidney, often shows marked vascular change as a result of hypertension. Thus, after a variable period of time, the kidney with the narrowed vessels, since it is protected from the hypertension it produces, becomes the good kidney. In this type of case, every attempt should be made to reconstruct the renal artery to save the renin-producing good kidney.

The case presented here demonstrates the precipitous fall that can occur after surgery. We feel that preparation should be made before surgery to maintain the blood pressure by intravenous pressor substances in the event of a dramatic fall. The marked lability of blood pressure that occurred in this patient postoperatively when norepinephrine was used as the pressor agent is interesting. Possibly, the conditioning of this patient's vascular tree from the hypertension of renal origin would make his vessels hyperreactive to norepinephrine.

A variety of methods of surgical management is available in treating unilateral renal artery disease. Reconstructive surgery of the right renal artery is difficult because the artery is tightly hemmed in by the vena cava and the renal veins. Therefore, as in the reported case, a right nephrectomy is a safer and more reliable operation to effect cure of the hypertension, assuming, of course, that left kidney function is known to be normal. The left renal artery is anatomically more accessible and therefore lends itself more to reconstructive methods. Endarterectomy for segmental obstruction or a plaque at the orifice is feasible. Resection of an offending segment and anastomosis can be done, or the renal artery can be reimplanted into the aorta at a more suitable site. At least one case wherein the splenic artery was brought down and anastomosed to the left renal artery has been reported. Arterial grafts can be useful. In the event of normal function in the contralateral kidney, the procedure on the right side will almost always be nephrectomy; on the left side, reconstructive methods

can be applied more commonly. If function in the contralateral kidney is abnormal, one is virtually forced to use a plastic reconstructive procedure, so as not to sacrifice good kidney tissue. The choice of procedure may represent a difficult decision, for example, with cases wherein the hypertension is of long duration and pathologic changes are suspected of having started in the contralateral good kidney as a result of the effects of the hypertension. Perhaps biopsy of the good kidney would be helpful under certain circumstances.

Previous examples of narrowing of the renal artery due to trauma, both acute and of long standing, have been presented in the literature. In this case, it is interesting to observe that narrowing of the right renal artery took place at the exact level of this patient's previously fractured vertebra and that, microscopically, the stenosis of the vessel did not show the characteristic findings of arteriosclerosis. Certainly, it is not unreasonable to assume a possible etiologic relationship between the trauma and the renal artery lesion due to gradual fibrosis in the vessel precipitated by injury without penetration of the vessel wall.

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Thomas A. Peppard, M.D.

HENRY L. ULRICH, M.D.

Minneapolis

IT IS WITH great pleasure that I write a short sketch of Dr. T. A. Peppard's activities in Minneapolis.

Dr. Thomas Albert Peppard was born in 1887. His forebears were of good European stock. He was educated in the public schools of Minneapolis. He entered the University of Minnesota Medical School in 1906 and graduated in 1911. After a year of internship at the Ancker Hospital in St. Paul, he received his degree of M.D. and also was awarded the Rollins Cutts medal—a prize in surgery.

In 1913, he was appointed assistant in the University of Minnesota dispensary in medicine and gynecology. The following year he "apprenticed" himself in the office of Dr. George Douglas Head, who was one of the leading internists in Minneapolis. After a year of this searching to find himself, he repaired to Devils Lake, North Dakota, as a general practitioner. In the two years which he spent in this community, his training and courage were put to the most trying tests. In his Devils Lake sojourn, he realized that industry, honesty, and humility were the essentials needed to meet the task of medicine. He never swerved from this attitude in the subsequent years.

In 1917, he returned to Minneapolis to practice internal medicine; at the same time, he joined the medical staff of the Minneapolis General Hospital which then had two divisions—a city group and a University of Minnesota group. In 1918, he came over to the side of the University. Soon after that, the two groups were united into one division staffed by University appointments. My association with Dr. Peppard at the General Hospital began in 1918 and continued until 1937, at which time I was transferred back to the University Hospital. No one knows except the participants of that period of twenty years what difficulties were encountered. No one realizes except the men involved what a transformation was accomplished in the medical service. Dr. Peppard's faithful cooperation and the high ideals we set for ourselves made this service into a clinic with all the attributes of University standards.

During the struggle for university standards at the General Hospital, we were greatly helped by Dr. Walter List, who gladdened and lightened our efforts by his cooperation. Dr. List was the first superintendent of the General Hospital who had been trained by the United States Public Health Service. The significance of this growth of the medical wards under our tutelage has been more or less forgotten by the new generation in charge. Its present dynamic growth must be a great source of satisfaction to Dr. Peppard.

In 1924, Dr. Peppard paused to find himself again by studying at the feet of Drs. Bauer, Wenckebach, and Neumann—famous clinicians in Vienna. German medicine was in its ascendancy. It was customary in the early part of the twentieth century for Middle Western doctors to go to Vienna for a "brush up."

As a clinician, Dr. Peppard is most meticulous in his habits. In discussion, he is slow of speech but finished and accurate. His clinical records are so well prepared that they could be published without editing. He has published very little; he has been too busy teaching. Again, he had a natural reluctance to add to the tons of medical literature emanating from an avid press. For this resistance against the pressure of publications, we are grateful to him.

Dr. Peppard's second allegiance to medicine was exhibited in his efforts to improve and raise the standards of the Asbury Methodist Hospital in Minneapolis. For this meritorious job, he was awarded a plaque by the staff and hospital board in 1954. In 1955, he was awarded the St. Barnabas bowl. This bowl is a symbol of achievement. The recipient is chosen by the Hennepin County Medical Society as the doctor of the year.

Dr. Peppard emerged into the era of medicine when the emphasis on morphology (pathology) and bacteriology was transferred to physiology and chemistry with their biophysics and biochemistry. Then came the overwhelming mass of factual knowledge and equally overwhelming load of chemo-

therapy and antibiotics. The concepts of the natural history of disease were smothered in factual discussions. He, like others of the earlier period, tried to carry over some of the best of the old into the new. He never veered from the fundamental idea that the best study of disease is the human body at the bedside.

Throughout this deluge of papers with "suppositions," "probabilities," and "suggestions" as conclusions, he never swerved in his teaching of medicine at the bedside.

Let me put it in another way. In my eulogy of him at the time he was honored by the Department of Medicine at the University of Minnesota in 1957 and became an emeritus associate professor of medicine, I said:

"Now I come to a most valuable attribute in Dr. Peppard's make-up. He has walked through this medical world of ours, which is filled with gadgets and drenched with biochemical factualities, unscathed and unafraid. Let me illustrate what I mean by telling a story. I borrowed it from Dr. Compton who is fond of telling it.

"It seems Dr. Compton had a sister who lived in India. She hired a local electrician to do some wiring in her home. During the day, the man interrupted her frequently with questions regarding the job.

At last she turned on him and said: 'You know what I want. Use your common sense.' The Indian bowing low replied: 'Madam, common sense is a gift from God. I only have technical knowledge.' Yes, Dr. Peppard has a generous measure of this God-given faculty.

"He has used his common sense in his life as a citizen. He has used his common sense in his teaching capacity at the General Hospital. He has used his common sense in his private practice as a clinician.

"Long may he continue in his pilgrimage blessed by the many young men whose medical life he has influenced."

There is one other item in this clinician's make-up. He soon noted that his medical education had lacked the humanistic disciplines. This he quickly and thoroughly modified by reading history, biography, and literature. One of the flowers of this reading is a paper in which he discusses his doctor-patient relation with Dr. Oscar Ferkins. This paper has never been published. Having read it, I can testify that it is a literary gem. Its historical value has placed it in the library of the University of Minnesota.

This quiet, unassuming but resolute man has a unique place in the local history of medicine.

MOSTLY BECAUSE OF the increased use of psittacine birds as household pets, psittacosis is becoming more frequent. The disease not only occurs after exposure to psittacine birds; epidemics arise among workers handling infected chickens, ducks, pheasants, and turkeys.

Among 40 employees who processed and shipped turkeys from a farm in Texas, 24 had psittacosis. The most common features were headache, fever, myalgia, cough, and anorexia. In 22, the illness was slight and subsided spontaneously or shortly after tetracycline therapy was begun. The other 2 patients had clinical and laboratory signs of hepatitis with renal decompensation; post-mortem examination of the 1 patient who died revealed extreme interstitial pneumonitis with tracheobronchitis, acute toxic nephrosis, acute nonspecific reactive hepatitis, and multiple focal lymphocytic meningitis.

E. M. YOW, J. C. BRENNAN, J. PRESTON, and SAMUEL LEVY: The pathology of psittacosis. *Am. J. Med.* 55:739-749, 1959.

BOOK REVIEWS

Recent Progress in Oxytocin Research

B. BERDE, M.D., 1959. *Springfield, Ill.: Charles C Thomas. 84 pages. Illustrated. \$4.75.*

The result of most efforts to review the literature on a given subject is a hodge-podge of factual material artlessly fabricated and devoid of interconnecting links. Nevertheless, reviews of this sort are useful because they furnish guidance for and nourish the thoughts of the expert worker in the field. "Recent Progress in Oxytocin Research" is different. Anyone who has had basic courses in biology and chemistry can assimilate a good portion of its contents in one sitting, yet there is no dilution of scientific information. The author has achieved this balance most cogently with direct and simple language, excellent illustrative material (13 tables and 37 figures in an 84 page monograph), and orderly presentation.

Starting with a concise historic note, he presents in logical sequence selected data on chemical structure and biologic activity of oxytocin and related analogues, production and fate of oxytocin in the organism, and the physiologic function of oxytocin in labor and lactation. The final chapter deals largely with the action of oxytocin on transport of spermatozoa within the female reproductive tract and the possible role of the hormone in the regulation of renal hemodynamics and urinary excretion.

On the whole, the author reviews and discusses the recent literature objectively. There is a brief lapse when he concludes that valyl-oxytocin is a more potent oxytocic and is, therefore, superior to the natural hormone. This conclusion is based on meager data which can be challenged. Moreover, the term "potency" has many meanings and, even when used appropriately, does not necessarily signify "superiority." The converse, of course, is also true. In all fairness, it must be pointed out that this issue is only a minor one and should not dampen enthusiasm for "Recent Progress in Oxytocin Research." Those who read this book will want to read it again.

EDWARD J. CAFRUNY, M.D.
Ann Arbor, Michigan

Symposium on Pulmonary Ventilation

R. P. HARBORD, M.D., and R. WOOLMER, 1959. *Baltimore: Williams & Wilkins Co. 109 pages. \$4.00.*

This volume records the proceedings of a symposium on Pulmonary Ventilation held in England under the auspices of the *British Journal of Anaesthesia*. The high quality of the material presented reflects the distinguished list of speakers. While the symposium was primarily directed toward the anesthesiologist and the pulmonary physiologist, the subject matter is of interest to all physicians concerned with the management of pulmonary insufficiency.

Perhaps the most valuable section is a paper by Dr. P. Hugh-Jones on the "Management of Pulmonary Ventilation in Emphysematous Subjects in the State of Carbon

Dioxide Narcosis." He assesses the proper role of oxygen therapy in obstructive emphysema and describes the methods of artificial ventilation with a cuffed endotracheal tube through a tracheostomy. Excellent graphs illustrate a description of the relationship of alveolar hypoventilation and ventilation-perfusion imbalance to the development of hypoxia and hypercapnia.

Although most authorities in this country would not agree, Dr. Hugh-Jones holds that chemical stimulation of the respiratory center with nikethamide or amphetamine is usually effective in early carbon dioxide narcosis. Unfortunately, documentation is not available to support this view.

Two papers deal with the differences in the partial pressure of carbon dioxide in end-expiratory air and in arterial blood. This gradient averages 5.3 mm. Hg in the anesthetized patient and may be explained by the development of a parallel respiratory dead space. Similar gradients could exist in disease, and thus the inaccuracy of equating end-expiratory carbon dioxide tensions with arterial blood tensions is emphasized.

A stimulating paper by Dr. E. J. Moran Campbell deals with the expiratory pressure-flow characteristics of patients with asthma and with emphysema. While airway obstruction is common to both conditions, this study suggests that different physiologic mechanisms may be involved.

This book, together with the appended references, is an excellent summary of current views on pulmonary ventilation.

STEPHEN M. AYRES, M.D.
New York

Diseases of the Chest Including the Heart

J. ARTHUR MYERS, M.D., PH.D., *Editor. Springfield, Ill.: Charles C Thomas. 1015 pages. Illustrated. \$34.50.*

This book is a collection of papers by 34 authors, each an authority on his subject. The time has arrived when such an assembly of currently useful information that has accumulated but was heretofore not readily available should be presented. The book is a large one because it covers the subject extensively.

It is printed on good paper and is easily read. There is a subject index and an author index. Each author has documented the material with an extensive bibliography, which adds further value to the exposition of the material presented. This one book so thoroughly covers the field that a collection of smaller books is less desirable than this one.

There is a large group of physicians who might not at first recognize how helpful this text might be to them unless they examine it. Anesthesiologists will find nothing directed at them, yet many of the problems they face are made understandable by these authors. This experience will be shared by most readers.

JOHN S. LUNDY, M.D.
Chicago

The Journal Lancel

SERVING THE MEDICAL PROFESSION OF MINNESOTA,
NORTH DAKOTA, SOUTH DAKOTA AND MONTANA

Series on OPHTHALMOLOGY

Ocular Therapeutics in General Practice

HOWARD A. SHAW, M.D.

Minneapolis

ONE OF 5 CONDITIONS usually brings the patient to the ophthalmologist for eye care: (1) discomfort in or about his eyes, (2) visual disturbances, (3) headaches, (4) abnormal positioning of the eyes, and (5) trauma. As is true in all other fields of medicine, therapeutic approaches follow diagnostic attempts. They are so entwined that separation is impossible. Certain tools, therefore, become essential in diagnosis.

The ophthalmoscope. This instrument will frequently need adjustment. If the view is still dull after new batteries have been inserted, the bulb should be examined. If it is blackened, a new bulb will give a clear image. A scope with a pin-hole aperture is helpful for looking through small pupils. The Kccler ophthalmoscope has such an aperture. The new May ophthalmoscope seems very satisfactory.

The loupe. This lens will be of service in many areas other than the eye. A very satisfactory loupe dispensed by optical companies, called the French Magnocular, can be purchased through local opticians for approximately \$27.50. This is collapsible so that it can be put in the lapel pocket and always be available. It fits over glasses easily. It magnifies 1.2 times and has a focal length of 8 in. A good-sized field is obtained.

Focal light source. The Welch Allyn pocket flashlight with a slit-focusing adaptor costs \$8.50

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but is invaluable, as the focuser allows quick judgment of anterior chamber depth.

Vision testing charts. A clean distance chart costs but 60 cents. When it becomes dirty it should be replaced. A real help is the reduced Snellen reading card to use with home or hospitalized patients. With reading glasses, the patient's visual acuity level is easily determined by this hand card.

The tonometer. This instrument should not be ignored. A good practitioner can easily learn its use, and an ophthalmologist will gladly demonstrate it. Most "silent" glaucomas will be detected by this instrument. The Schiøtz standardized model is \$35. Remember that 2 per cent of patients have undetected glaucoma. Detection is the general physician's responsibility in a routine physical.

Fluorescein. This solution is easily contaminated. Fluor I strips—sterile fluorescein applicators that look like Band-Aids—are available. A drop of sterile water is placed on the end of the strip after it is removed from the sterile envelope, and this is placed on the conjunctival side of the lower lid. This is convenient, easy, and safe. They may be ordered in boxes of 200 and cost 2½ cents apiece.

The scope of this paper does not permit mention of all the various useful drugs. The introduction of the plastic squeeze bottle has been very helpful in eye care. No dropper is necessary, thus helping to avoid contamination. The unit is heat-sealed also, helping to preserve sterility.

A few essential items that should be conveniently placed on the office treatment tray are:

1. Ophthaine solution, 0.5 per cent, which is a satisfactory topical anesthetic. Dosage is 2 drops, repeated in 15 seconds. Do not drop on the cornea so as to avoid a blinking reflex.
2. An atomizer squeeze bottle with a sterile aqueous solution (normal saline or boric).
3. Elastoplast, 2 in. width.
4. Oval eye pads in a sterile envelope.
5. Fluor I strips.
6. Sodium Sulamyd ophthalmic ointment (10 per cent), 1 tube.
7. Polysporin ophthalmic ointment, 1 tube (polymyxin B sulfate and zinc bacitracin).
8. Neo-Synephrine, 10 per cent, in a 5-cc. monodrop bottle.
9. Isopto-carpine, 1 per cent, in a 15-cc. droptainer.
10. Scotch tape dispenser, ½ in. wide.
11. Atropine solution, 1 per cent, in a red-topped dispensing plastic bottle.
12. Cyclogel, 1 per cent.
13. Neosporin ophthalmic solution, 0.1 per cent, 5 cc.
14. NeoDecadron ophthalmic solution, 0.1 per cent, 5 cc. (dexamethasone 21 with neomycin).

CARE OF THE PATIENT

Foreign body sensation. Two drops of Ophthaine are instilled first to relieve blepharospasm. Loupe and focal light detect foreign bodies which, if found, should be dislodged by a stream of saline. If they remain, they should be removed with sterile golf club spud. One must be sure to get under the foreign body to lift it out; otherwise only the top of the foreign body may come off.

Rust rings. These are common and difficult to remove because the corneal cells have taken up the iron. The patient should keep both eyes open and fixed on some predetermined object. The rust bed is scraped very gently and not deeply. An ointment dressing and eye pad are then applied and secured with Elastoplast. The wound should be observed daily until there is no staining with fluorescein. If the physician is shaky and the rust ring is not easily handled, he may prefer to leave it rather than traumatize the cornea. After five days, liquefaction of the cells occurs and the ring may be lifted out easily.

If no foreign body is seen on the cornea, the patient should look *down* while the physician everts the upper lid. A cotton applicator is used

to lift the foreign body from the conjunctiva at the upper edge of the tarsus where it usually lodges, and the cornea is stained. If numerous abrasions are present, an ointment is put on the eye, which is bandaged for twenty-four hours. If no foreign body is found, the cornea may take stains showing tracks left by a previous foreign body. Look carefully into the punctum to be sure that a cilium has not lodged there. If still no foreign body is found and one is dealing with a menopausal woman, he may find that she has decreased tear flow, producing epithelial erosion. Tears may be checked by a piece of filter paper cut 35 mm. long and 5 mm. wide and folded over the lower lid at the lateral side. Within five minutes, the strips should be completely wet with lacrimal activity. If there is no evidence of tearing, the patient is probably not lubricating the eyeball and considerable relief can be obtained for her with Tearisol, an artificial tear and lubricant.

Frequently, physicians overlook spastic intermittent entropion as a cause for a foreign body sensation. The patient should close his eyes tightly, allowing the physician to see if the lashes invert onto the cornea. Tranquilizers may be used temporarily with the lid taped down, but usually a surgical procedure is necessary to relieve this condition.

Welding flash burns. Usually these burns produce symptoms about eight hours after exposure to the flash. Therefore, the physician is aroused from sleep to care for the patient. The lids are edematous, and punctate erosions of both corneas are present. A sedative and analgesic are necessary as well as a topical corneal anesthetic, such as Butyn and Metaphen ophthalmic ointment or Pontocaine. Both eyes should be tightly covered and the patient given reassurance that he will recover his vision. It is wise to leave a sedative at the house so that the patient will not call again during the night.

Corneal ulcers. These are extremely difficult to manage and require considerable care. They may lead to serious ocular impairment.

1. *The dendritic ulcer* (herpes simplex) can be exceedingly stubborn. It is usually near the center of the cornea and stains as an arborizing branch with little knobs. Experience has shown that cortisone derivatives must *not* be used. Extension of the ulcer or perforation in the anterior chamber may occur. Treatment involves (1) anesthetizing the eye with Ophthaine topically, (2) applying full strength iodine to the ulcer on a firmly bound cotton toothpick applicator, (3) neutralizing with 4 per cent cocaine, (4) bandaging tightly after applying 5 per cent ho-

matropine drops and an antibiotic ointment, and (5) dressing in forty-eight hours. If difficulty continues, the patient should be referred to an ophthalmologist.

2. *In herpes zoster ophthalmicus*, corneal anesthesia is usually present. This can be checked for with a small piece of cotton. If any of the lesions usually seen on the forehead and face are present on the tip of the nose, the nasociliary nerve is involved. Involvement of the eyeball frequently takes place after this occurs. Care of the eye consists of protecting it while in the anesthetic stage and dilating the pupil if a corneal ulcer occurs. Care of the eye consists of protecting it while in the anesthetic stage and dilating the pupil if a corneal ulcer occurs. Many therapeutic measures have been given for the treatment of this disease. A practical one might be the daily use of vitamin B₁₂ intramuscularly plus the use of steroids to relieve the pain and to cut the edema surrounding the gasserian ganglion. Protamide, 1.3 cc. intramuscularly daily for four days, and convalescent serum have been used, but their efficacy is questionable. The eye may be seriously involved if the cornea has a deep-seated lesion. As mentioned, the eye should be protected from trauma from outside sources and atropine should be applied daily. Encephalitis occurs at times, and steroids increase mortality.

3. *Marginal ulcers*. These lesions frequently form on the cornea secondary to an acute conjunctivitis and will heal as the conjunctivitis heals. There is usually a small separation at the limbal margin of the ulcer from the scleral side. I do not bandage these eyes while treating the conjunctivitis. *Pseudomonas* is especially virulent to the cornea; it produces a greenish-colored ulcer and needs immediate treatment with polymyxin and neomycin. It is a common contaminant of fluorescein solution and, as we mentioned, is the reason why we prefer sterile fluorescein strips.

4. *Epidemic keratoconjunctivitis*. This disease is a virus involvement leading to punctate keratitis, which may annoy the patient for months. He is made more comfortable if he uses a cycloplegic, such as 2 per cent homatropine, every other day and wears dark glasses. Reassurance that he will eventually be all right is important. The use of the tonometer without sterilizing the foot plate can be a common source of contamination of eyes with this virus. The eyes should not be overtreated.

Lid infections. A styte is usually painful and a chalazion is not. Hot packs and an antibiotic

ointment will lead to localization of both. The styte may then be drained. The chalazion should be evacuated after five to seven days. This is accomplished relatively easily with the following technics: (1) anesthetize the eye with Ophthaine, (2) apply Ophthaine to a cotton applicator and rub on the conjunctiva over the chalazion, (3) use a dental syringe with a 30-gauge needle and cartridges of Novocain to inject the palpebral conjunctiva just above the chalazion, (4) after a five-minute wait, apply chalazion clamp, (5) incise palpebral conjunctiva vertically over the chalazion on the firm tarsal bed, (6) express the chalazial contents with a toothpick cotton applicator, (7) apply an eye ointment and pad, and (8) instruct the patient to remove the pad upon returning home and to use an ointment three times a day for two days with hot applications, then twice a day for three days.

Acute conjunctivitis. This condition may be treated empirically, and the eye is not bandaged. Drops are used during the day and an ointment at night. In the presence of pus, the sulfonamides may not be effective. If no improvement occurs within three days, medication should be changed. If the conjunctivitis has not cleared in six days, a culture and smear should be taken and the sensitivity reports checked.

Ophthalmia neonatorum. After immediate culture and smear, penicillin drops should be started—3,000 units per cubic centimeter every minute for one-half hour, then every five minutes for one-half hour, then half-hourly, then hourly for two days. The other eye can be protected by a shield of exposed x-ray film.

Chronic blepharitis squamosa. This is a common affliction of adults and children. It is closely related to seborrheic dermatitis. The lids are treated by (1) removing all crusts from the margin thoroughly with cotton and tap water, (2) patting dry with a dry cloth, and (3) rubbing Metimyd ophthalmic ointment into the base of the lashes at bedtime for ten nights.

In addition to this therapy, care of the scalp is essential, and care of the eyebrows might be included. The use of Selsun or other similar derivatives is helpful in maintaining hygiene of the hair. Unless this is done, the condition of the eyelids usually does not improve. Chronic recurrences are common, and brief treatment with the ointment may be necessary.

Allergic conjunctivitis. NeoDecadron drops, 0.1 per cent, are administered to the eyes five times daily. The skin is managed as in any allergic manifestation. Cold saline compresses to the face are used in the acute phase and soap prepa-

rations are not used. Teldrin, 12 mg. twice a day, is used for the antihistaminic effect. Tranquilizers may be given, if necessary, but not barbiturates. Hydrocortisone, 1 per cent, in eye ointments is administered in subacute stages. Every effort must be made to find the offending source. Cosmetics and pollens are common factors around the eye. If penicillin is the cause of dermatitis medicamentosa, 800,000 units of penicillinase (Neutrapen) in 2 cc. of aqueous solution, should be given intramuscularly.

Visual disturbances. Visual acuities should be recorded; an accurate history should be obtained, ascertaining whether acute or gradual change in vision occurred and whether pain was present. The state of the cornea, lens, vitreous, and retina should be observed.

SUDDEN VISUAL LOSSES

Glaucoma. This disease should be suspected if the cornea is edematous or steamy. If glaucoma is present, the pupil will be dilated, the eye red, and the patient in acute distress and, perhaps, vomiting. The eye will be hard, and the patient will need ophthalmologic help. The general practitioner can aid by giving emergency treatment of $\frac{1}{2}$ gm. of morphine sulfate and a good diuretic. Eye drops should *not* be used until the ophthalmologist has seen the patient. Diamox, 500 mg., may be given intravenously without harm if the patient is vomiting. If he is not, Diamox or 50 mg. of Daranide may be given orally. Urea, 30 per cent in invert sugars, is a potent decompressor. The ophthalmologist will undoubtedly operate on a patient with glaucoma when he has reduced the tension.

Occlusion of the central retinal artery. This condition demands immediate therapy, and the patient should not be transported to an ophthalmologist unless one is immediately available. The patient should be given (1) amyl nitrite inhalation, (2) 500 mg. of Diamox intravenously to reduce intraocular pressure and thus increase effective vascular pressure, (3) inhalation of CO₂ by having the patient breathe in a bag to improve retinal vascular flow with the higher CO₂ concentrations, (4) 2 cc. of Hydergine, intravenously or intramuscularly, and then 1 tablet sublingually four times a day, (5) oxygen administration, after the initial therapy and the CO₂, by either nasal catheter or mask inhalation at bedside.

Use of anticoagulants is problematic. I see no contraindication to heparinization and maintenance on anticoagulants for a considerable time. The ophthalmologist is of no help other than in interpretation of the original diagnosis.

The appearance of the fundus showing a whitish, edematous background with a red macular area and a boxcar-like effect in the retinal vessel circulation will certainly aid in the diagnosis.

Central or tributary retinal vein thrombosis. Treatment with anticoagulants is a must. Heparin should be administered intensively for ten days to three weeks. With sclerotic patients, this therapy can be continued for a longer period. Vitamin E, 100 mg. three times a day, is also continued indefinitely. Late glaucoma development is thus reduced, and visual function is possibly restored. A careful evaluation of the serum proteins of the patient should be done for the possibility of albumin-globulin ratio changes.

Acute iritis. In these cases, the cornea is lusterless, keratic precipitates appear on the inferior surface of the cornea, the pupil is miotic, and the iris appears dull. The vitreous may seem quite hazy, and numerous cells may seem to be moving about. Pain, photophobia, and tearing accompanied by visual loss are usually present. The ophthalmologist is primarily concerned with maintaining ocular function by preventing adhesion of the iris to the lens (posterior synechia) while the disease runs its course. He therefore dilates the pupil widely with atropine and Neo-Synephrine. Two main classifications of iritis occur: serous and granulomatous.

1. The serous is the more common type and is seen with rheumatoid arthritis. Usually, the disease runs its course, but a search for foci of infection, such as the teeth, pelvis, rectum, and pharynx, is necessary. Triple typhoid is a potent weapon as a foreign protein and is given intravenously by the ophthalmologist every other day for five treatments. Steroids may be helpful. Desensitization to streptococcal allergens may become necessary.

2. The granulomatous lesions are treated specifically. In possible tuberculous nveitis, steroids are avoided.

Optic neuritis. Sudden visual reduction, accompanied by pain on turning the eyes, is indicative of optic neuritis. One looks for several possible primary causes, such as diabetes mellitus, multiple sclerosis, and toxins.

Optic neuritis is treated with daily intramuscular injections of vitamin B₁₂. Steroids may also be given parenterally to reduce the edema, which produces optic nerve atrophy.

Temporal arteritis. Severe headache and reduced vision occur in this disease. Steroids are essential to prevent blindness. Involvement of the other ophthalmic artery will also occur if treatment is not instituted. The temporal artery can be felt to be enlarged and attached to the

underlying skin and is severely tender to touch. A biopsy of this artery will confirm the diagnosis.

Vitreous and retinal hemorrhages. These lesions may obscure underlying retinal detachment or a tumor. These are serious problems requiring ophthalmologic help. I shall not deal with the hypertensive arteriosclerotic retina or that of the diabetic retina, as the management of these rests in the fields of medicine. We have no help at the present for the prevention of diabetic retinopathy.

PAINLESS LOSS OF VISION

Glaucoma. The deep chamber or chronic simple type of glaucoma is insidious in onset. Tonometry is essential. One must detect an elevation of tension before the optic nerve head changes occur. If any tension of 25 mm. Hg or more on three repeated visits is found, the patient should be referred to an ophthalmologist. The eye is white, unlike the acute glaucomatous eye.

Transitory myopia. This condition should be checked carefully for diabetes.

Pigmentary macular changes. These are extremely common and are the nemeses of aging. They are vascular in nature. Anticoagulants at the present time, however, appear to offer a form of therapy that may possibly be of help in the future. With the use of anticoagulants, some edema occurs, but this gradually diminishes and visual acuity may improve.

Retinal detachment. The retina appears slate-gray and the vessels appear thread-like black. This type of problem needs ophthalmologic help at once because of the possibilities of an underlying tumor. If a retinal tear is present, retinal reattachment with diathermy is done. Retinal detachments which occur in pregnancy with a hyperemesis gravidarum do not need surgery.

Hysteria. This phenomenon is not uncommon, and tubular vision is usually noted in these young patients. *Conical corneas.* These develop in young adults and are greatly benefited by contact lenses. The retina appears blurry with the ophthalmoscope.

Papilledema. In these cases, there is a late loss of visual acuity and one must rule out intracranial pathology. The raised optic nerve head can be measured with the ophthalmoscope and some distention of the returning venous flow as well as hemorrhages around the optic nerve head itself can be seen.

INJURIES

Intraocular foreign bodies. If the patient has been hammering and feels something strike his eye and nothing can be seen, one must be suspi-

cious. Anteroposterior and lateral roentgenograms of the eye must be made. One must be careful of artifacts on the screen. If a foreign body is found and the radiologist can do a Sweet localization, it may be determined whether the foreign body is in the globe or in the orbit. A foreign body left within an eye will usually destroy it. The pupil should be dilated for a good survey of the vitreous and retina. Referral is usually necessary.

Chemical burns. Immediate dilution of the offending agent is essential. The patient should not be transported to a doctor's office. Trained personnel at plants should have the patient put his head and eye under flowing water for at least five minutes before he leaves. If it is an acid burn, all the damage will have been done quickly. If it is a solid alkali, the particles must be removed from the eye or continuous hydrolysis will take place. The eye may be anesthetized with Ophthaine for careful examination with a loupe. After thorough irrigation, 0.1 per cent of NeoDecadron drops should be given every half hour for two hours, then every hour for two days. The eye must be atropinized. A bandage makes the patient more comfortable, and dark glasses should be worn. Sedation is desirable. We do not neutralize the chemicals any more, as we feel the heat of neutralization creates additional damage to the cornea. If the lids are burned, the conjunctiva must be freed daily from the globe to prevent symblepharon from occurring. A sterile glass rod will suffice for this purpose.

Hyphema. This condition is always potentially serious. A rigid pattern must be followed in its management. The bleeding is probably caused by rupture of the ciliary body following a blow to the eye.

No drops should be put into the eyes, because a dilated pupil may block access of blood to the chamber angle and decrease blood absorption through the iris bed. The patient should be given a sedative. Absolute bed rest with both eyes bandaged and the bed at a 15° elevation, so that the blood settles to the bottom of the cornea, is prescribed. Binocular occlusion reduces ciliary muscle reflex activity. Parenzyme, intramuscularly or sublingually four times a day, reduces the local inflammatory condition. This program should be maintained for five days. If the corneal reflex becomes black due to a massive hemorrhage into the anterior chamber, immediate ocular consultation should be held, as glaucoma is likely to ensue.

Rupture of the globe. The lids should not be pried open in a patient who is upset. Sedation

should be given, and then Ophthaine is dropped inside the lower lid. Soon the eye will be visible. If the chamber is collapsed and the iris is protruding, further investigation should be stopped. No other medication should be put in the eye. The eye should be bandaged gently and the patient transported to the hospital for a specialist's care.

Any underlying scleral rupture should be suspected also. The conjunctiva, if slightly torn, will heal spontaneously. If the gap is larger, it may be closed with 6-0 running silk. Anesthesia is administered topically and the conjunctival wound spread apart to obtain a good view of the sclera and determine whether a rupture exists.

Lid damage. If such damage is severe in the inner canthal areas, the canaliculi will be torn. These are difficult to repair and, if not repaired successfully, constant tearing occurs. When lid tears are approximated, a 6-0 suture through the cut on the free lid margin will prevent notching. Polyethylene tubing may be placed across a torn canaliculus and into the nasolacrimal sac and duct.

PEDIATRIC EYE CARE

We have touched on some problems related to children. In general, if a child's eyes are turned at 4 months, they should be carefully scrutinized for congenital glaucoma, cataract, or retinal damage, especially that from retinoblastoma or toxoplasmosis. The eyes should be dilated with 10 per cent Neo-Synephrine. While the mother is feeding the child, the doctor can look carefully at his eyes. If he has congenital glaucoma, the eye will appear large and look like an ox's eye. Immediate surgery of the chamber angle by goniotomy is necessary to save his sight.

If a *cataract* exists, surgery is dependent on the vision likely to be present in the two eyes. If both eyes are seriously involved, a cataract operation should be done at 1 year of age. A monocular cataract may well be left alone. If visual abilities in the two eyes are estimated to be about 20/60, then surgery should be deferred.

A *retinoblastoma* will be seen as a mass in the vitreous producing a yellow reflex. Immediate referral is necessary, as it may be present in the other eye also. At this time, if the eye is seriously involved, it will be enucleated, and, if further retinal changes are present in the remaining eye, this eye is treated with radiation and triethylene melamine.

If *strabismus* exists, all eyes should be examined from the age of 4 months and after for the reasons given in the preceding paragraph. All

care relative to the child should be completed by the time he is ready to enter kindergarten. Glasses are supplied if necessary, and occlusion of the fixing eye to improve the vision of the suppressing eye may have to be done. If necessary, surgery and orthoptics follow. The psychological influence of a crossed eye on a child going to school is quite marked.

Retrolental fibroplasia. A definite response of the retina to excessive use of oxygen in the premature infant produces a fibroplastic proliferation of the retinal vasculature. Only that amount of oxygen compatible with life should be used. If more is used, a careful scrutiny of the fundi is essential, as the process is reversible in an early stage. The findings should present themselves within the first 4 weeks of life.

Obstruction of tear ducts (epiphora of the new born) is best treated with an antibiotic ointment, and the mother should massage the nasolacrimal sac from the top to the bottom daily for at least ten days. She may then observe the status of the eye and whether a purulent discharge continues. If it recurs, she may repeat the process again. If no improvement occurs after ten months, a probing of the nasolacrimal duct will be necessary. Fifty per cent of all nasolacrimal duct obstructions will spontaneously open at the end of ten months.

DIPLOPIA

Diplopia is always viewed by the ophthalmologist as a serious problem. If it is sudden in a young adult, he will think of multiple sclerosis. If accompanied by severe pain and ptosis, he thinks of the possibility of an intracranial aneurysm and listens for a bruit over the eyeball. If it occurs in a diabetic, diabetic neuropathy is likely. If the movements of the two eyes are equal in excursion but one is turned and the other is fixing, an upper motor neuron involvement, probably vascular or neoplastic in nature, is suspected. Annoying, constant diplopia can be avoided for the patient by alternately covering one eye. If corneal anesthesia is down, one must think of an acoustic neuroma. This is present also as a prodromal symptom in developing herpes zoster. If the diplopia occurs posttraumatically, a complete or partial recovery may occur in one year. No definite surgery should be done before that time. The postthyroidectomy patient may go on to further exophthalmos. In this case, permanent changes occur in the muscle structures, making control of the diplopia extremely difficult. At a late date, it may be necessary to do surgery for cosmetic purposes. Prisms may help this patient but usually do not.

RECENT ADVANCES

The most recent exciting therapeutic advance in ophthalmology has been the introduction of alpha chymotrypsin. This enzyme allows the lens to be removed more easily in cataract surgery because it dissolves the zonules holding the lens in place. It does not dissolve the lens itself, which must be removed as it was before in cataract surgery. It is not used in children under 15. The addition of the carbonic anhydrase inhibitors, such as Diamox, during the past few years has greatly advanced our therapy in the

glaucomas. An exciting adventure in the trace of retrolental fibroplasia was done by men in the ophthalmologic field who finally discovered the cause to be that of excess oxygen, as has been previously mentioned. This tremendous contribution to medicine has greatly reduced the number of potentially blind children in our country.

All ophthalmologists feel that they would like to encourage the general practitioner to consider the use of the tonometer. The ultimate detection of glaucoma cases rests in his hands.

LOCAL TISSUE hypersensitivity to catgut may produce eye discomfort and swelling after eye muscle operations. The eye itches, and inspection reveals chemosis and hyperemia of the conjunctiva and edema of the eyelid. In patients who have had previous eye operations, an intense reaction may appear within twenty-four hours, persisting a week if untreated. Usually, symptoms appear after a week and subside within twelve to forty-eight hours. Outcome of operation is not affected by the reaction.

Moist compresses, oral antihistamines, and locally applied steroids are used to treat delayed reactions. Cold compresses and locally applied steroids soothe and shorten immediate reactions.

Eye reaction and response to a small piece of catgut buried intradermally on the volar forearm were compared in 219 children having eye muscle operations. Plain catgut 4-0 sutures rinsed in sterile physiologic saline solution were used. No local eye medication was employed postoperatively. Positive eye and skin reactions appeared in 70 children. The reactions occurred within twenty-four hours in 13 children; all early reactors had had previous eye operations. The reactions appeared within five to nine days in 57 patients; 11 late reactors had had previous eye operations. Results of the skin test were negative in 4 patients with positive eye reactions; the abnormal appearance of the eye could be attributed to wound infection in 2 and extensive surgery in 2.

L. APT, F. D. COSTENBADER, M. M. PARKS, and D. G. ALBERT: Catgut allergy in eye muscle surgery. *Arch. Ophthalm.* 63:30-35, 1960.

Ophthalmic Facies

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THE PATIENT who confronts us in the office may, by his countenance alone, convey a diagnosis. A complete work-up is a must for correct diagnosis and therapy. However, in ophthalmology, as in other fields of medicine, the "coup d'oeil" is the essential part of our findings. It is the purpose of this brief review to summarize the more common physiognomy with relation to ocular disorders.

A triangular notching of the upper lid, usually at the junction of the inner and middle third, is indicative of a coloboma. The defect may vary from a slight indentation to a more complete nicking of the lid thickness. Lashes and glands are absent in the affected area. Plastic procedures are often indicated to prevent damage to the cornea from exposure.

Drooping of the upper lid, due either to a weakness or absence of the levator muscle, is diagnostic of ptosis. The degree of ptosis may vary from a slight narrowing of the palpebral fissure to a complete relaxation of the upper lid, which then hangs down, occluding the pupil. A typical facies, with the child throwing the head back or wrinkling the frontalis muscle in an attempt to open the eye, is present. Correction of ptosis by utilizing the superior rectus muscle, resection of the levator, or creating a "sling" with the lid attached to the frontalis muscle can lead to marked cosmetic improvement.

In many cases, epicanthus is associated with ptosis. The condition may be bilateral. A vertical fold of skin extending from the base of the nose to the inner end of the brow, concealing the inner canthus and caruncle, is diagnostic of this condition. In Mongolians, it is a racial feature. In Caucasian children with a broad flat nose and widely separated eyes, a false diagnosis of convergent strabismus is common. Pinching of the skin at the base of the nose corrects the false impression in milder cases, and surgery can be used to correct the more marked deformities.

Other congenital deformities, in which closure of the fetal fissures occurs too early, can give

the typical appearance of oxycephaly, Crouzon's disease, or hypertelorism. Abnormal development of the orbits causes proptosis, and stretching of the optic nerve can lead to papilledema and optic atrophy, with resulting defective vision.

Inflammatory lesions of the lids are hordeolum, chalazion, meibomitis, trichiasis, blepharitis, and dacryocystitis.

Hordeolum is an acute inflammation of the edge of the lid accompanied by pain, tenderness, and considerable edema. It is due to a staphylococcal infection of the glands of Zeis or Moll. Soon, one area of the lid becomes more swollen, and a yellowish point forms and ruptures spontaneously. Warm, moist compresses are best used in these cases to hasten suppuration.

Chalazion is an enlargement of one of the meibomian glands (figure 1). A stoppage of the gland occurs, causing either quiet or inflammatory "lump-like" engorgement of the gland. Size may vary from only a few millimeters to as large as 10 mm. These lesions are circumscribed and adhere to the tarsal plate, not to the skin. Treatment with hot packs alone is often sufficient. The small majority, 15 per cent, that do not disappear are opened on the conjunctival side, everted, and cauterized at the base with trichloroacetic acid or diathermy.

Misdirected lashes may cause irritability and congestion of the globe, photophobia, and lacrimation. Trichiasis can best be cured by epilating the aberrant lashes. Distichiasis, in which the row of meibomian glands is replaced by a row of lashes, is usually corrected by surgery.

The patient who seeks our help for reddened, scaly lid margins is an all too frequent one. The dry, scaling lid margins, with only a slight reddening of the area, may have the yeast-like organism, *Pityrosporum ovale*, as an etiologic agent. The whitish scales at the root of the lash follicles cause the lashes to fall out without destruction of the follicle itself. The ulcerative variety of marginal blepharitis is characterized by reddened, swollen lid margins with yellowish

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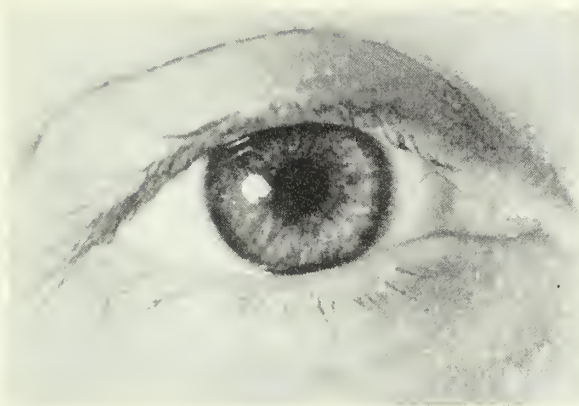


Fig. 1. Chalazion. Note enlargement of meibomian gland.



Fig. 2. Acute dacryocystitis. Swelling is seen at the inner canthus, just below the medial canthal ligament.

crusts, causing the lashes to adhere and leading to their deformity or destruction. In both forms, the patient will experience soreness, epiphora, itching, ocular fatigue, sensitivity to light, and sticky lids on arising in the morning. Conjunctivitis may accompany the blepharitis. Complications include sties, loss of the lashes, trichiasis,

hypertrophy of the lid margins, and ectropion.

Treatment consists of improvement in personal hygiene, thorough cleansing of the lid margins, and application of an antibiotic to which the staphylococcus is sensitive. The treatment is often very difficult, and caution must be exercised in promising complete cure.

Dacryocystitis, or inflammation of the lacrimal sac, may appear as a swelling at the inner canthus just below the medial canthal ligament (figure 2). The inflammation may be acute or chronic. If blockage of the lacrimal apparatus occurs, epiphora may be included in the symptomatology.

Warm packs and antibiotics are the chief means of alleviating the discomfort. Local incision of the lacrimal sac is only rarely indicated. Chronic dacryocystitis is best treated by dacryocystorhinostomy.

The patient who presents with a bilateral or unilateral lid edema may have one of several disorders. If inflammatory in nature, the lids may be swollen due to sties, dacryocystitis, and affections of the nasal sinuses. Severe inflamma-

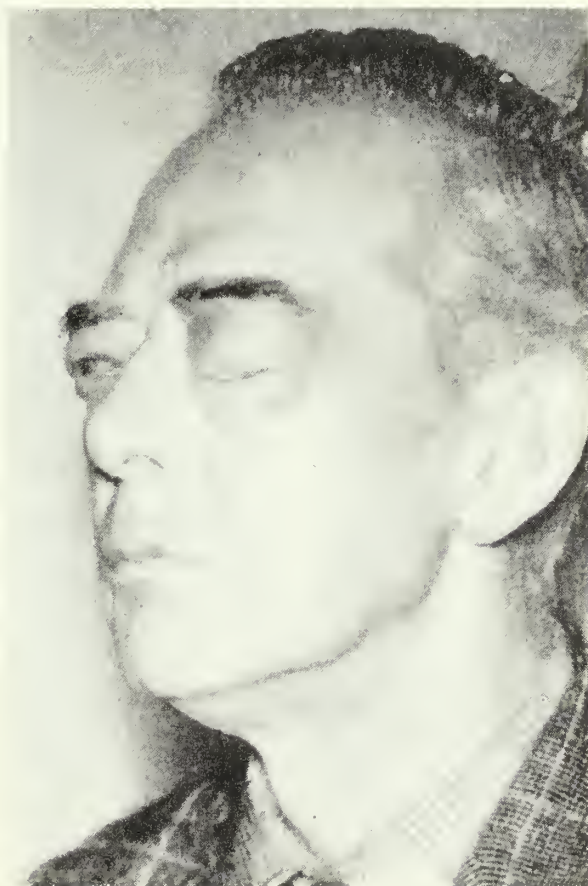


Fig. 3. Thyrotropic exophthalmos.

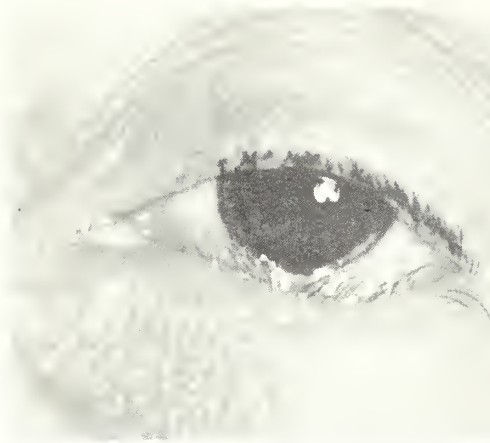


Fig. 4. Inward and outward rolling of the lid characterizes spastic entropion (*left*) and senile ectropion (*right*).

tions of the eye itself, such as acute glaucoma, iridocyclitis, orbital cellulitis, and panophthalmitis, may also cause generalized lid edema.

Traumatic events, insect bites, renal and cardiac systemic diseases, metabolic disorders (figure 3) and allergic substances are all etiologic agents of lid edema. Angioneurotic edema, which is recurrent and occurs rapidly, is most commonly seen in women at the time of their menstrual period. The edema may be severe enough to completely close the lids and generally abates spontaneously. Blepharospasm, with complete closure of the lids, is a puzzling entity treated by injection or surgical sectioning of the seventh nerve.

Dermatitis of the lids may be the result of local irritating agents, eye drops, poison sumac or poison ivy, and irritating conjunctival or lacrimal discharge. Discontinuance of the injurious substance will afford relief.

Herpes zoster ophthalmicus is characterized by a unilateral herpetic eruption along the path of the ophthalmic division of the fifth nerve. The patient usually notices a neuralgic pain on the affected side of the head and face, followed by a vesicular eruption which dries, leaving crusts and disfiguring scars. If the nasal branch of the nerve is involved, the eye becomes inflamed, leading to a diffuse keratitis and iridocyclitis. Treatment is purely symptomatic; for the facial dermatitis, mydriatics are instilled repeatedly in the affected eye.

Entropion, or the rolling inward of the margin of the lid, can cause injuries to the cornea and symptomatology consisting of pain, photophobia, congestion, and excessive tearing (figure 4a). Surgery is indicated for correction of this condition.

Ectropion, or the outward rolling of the lid

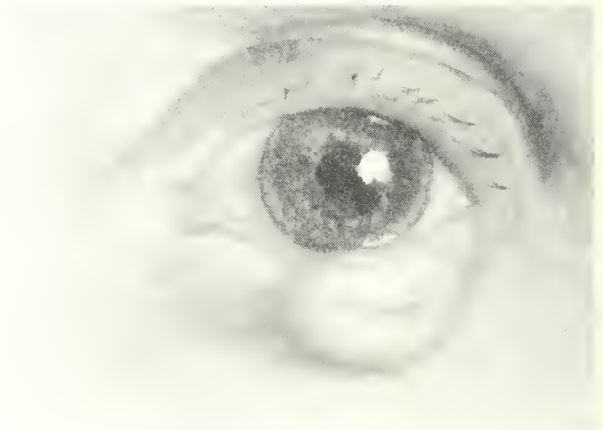


Fig. 5. Basal cell carcinoma on lid margin.

margin, is also corrected by surgical means (figure 4b). If left unattended, epiphora and drying of the conjunctiva and cornea as well as corneal ulceration can occur.

We have all seen the patient with a firm, oval swelling beneath the external canthus or beneath the medial portion of the upper lid. A dermoid is the common tumor found in this location and is easily removed surgically.

More extensive surgical procedures are indicated when a mixed tumor of the lacrimal gland is diagnosed as a cause of unilateral lid edema. These tumors are found beneath the lateral one-third of the upper lid. Orbital tumors, metabolic disorders, aneurysms, and inflammatory conditions must be considered in any patient with a proptosis or exophthalmos.

Diffuse port wine stain of one side of the face due to a capillary hemangioma is often accompanied by glaucoma on the affected side and intracranial hemangioma, as described by Sturge and Weber.

A disease marked by flushing of the nose and cheeks, together with a scaly condition of the lid margins and often corneal ulceration, is diagnostic of aene rosacea. Beta radiation has been used with some success in these cases.

Hematoma of the lids is disfiguring but causes no undue effects. Cold compresses applied within twenty-four hours of the trauma, followed by hot compresses, often hastens the absorption of blood which has diffused into the tissues.

Xanthelasma, the flat yellowish discoloration of the skin, usually at the inner canthus, is a collection of fatty degenerative cells and may be removed for cosmetic reasons.

Papillomas at the lid margins are benign and may be snipped off; milia, the small yellowish elevations at the lid margins, may be expressed, and small cysts which are obstructions of the glands at the lid margins are easily opened with a sharp needle or blade.

Basal cell carcinomas are round, indurated growths with rolled edges found chiefly on the lid margins (figure 5).

Squamous cell carcinomas are more diffuse in nature than the basal cell or rodent ulcer and may metastasize to adjacent lymph nodes. Both types of malignancy may be treated with radiation or surgically removed.

A FISTULA between the internal carotid artery and cavernous sinus most frequently causes impaired or blurred vision, always on the side of the lesion. Since slight diplopia may be confused with visual blurring, tests of visual acuity are not reliable. Objective signs may include a bruit, proptosis, conjunctival chemosis, periorbital flush, and venous dilation. Glaucoma is a frequent complication, often occurring after unsuccessful surgery in connection with cataract formation.

When a carotid-cavernous fistula is suspected, the following procedures are recommended:

- Repeated neurologic examinations
- Serial ophthalmologic examinations including funduscopy, visual field, and visual acuity studies and exophthalmometer, tonometer, and ophthalmodynamometer measurements
- Skull films in search of fracture
- Prophylactic tarsorrhaphy if extreme proptosis is seen with damage to trigeminal and facial nerves
- Bilateral carotid angiographic studies to determine patency of the anterior communicating artery
- The Matas test, with thirty-minute compression of the internal carotid artery before cervical ligation in young patients and gradual occlusion of the artery over several days in patients over 45

Recommended treatment includes combined cervical and intracranial internal carotid ligation with clipping of the ophthalmic artery.

J. W. HENDERSON and R. C. SCHNEIDER: Ocular findings in carotid-cavernous fistula in series of 17 cases. *Am. J. Ophth.* 48:585-597, 1959.

Headaches

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HEADACHES comprise one of the most common of all neurologic complaints. In large studies of unselected cases, the frequency has varied from 8 to 65 per cent. The incidence is greatest among females, young adults, unmarried individuals, students, executives, and members of professional groups. The cause of this complaint may vary from such a benign but distressing condition as tension to such a very serious illness as a brain tumor. Hence, this complaint often taxes the skill and diagnostic abilities of the physician. A careful history is often the single most useful tool in arriving at the correct diagnosis. However, the history only suggests the possible etiology and must be substantiated by careful physical examination and, if necessary, laboratory findings. It must be kept in mind that, although certain etiologic types of headaches produce suggestive histories, there is great overlap in complaints which can lead to error in the individual case.

MECHANISM OF PAIN

Contrary to what one would suspect, the brain and its coverings, the meninges, are insensitive to pain. The structures sensitive to pain within the intracranial cavity are the venous sinuses, the dural floor, the dural arteries, and the arteries at the base of the brain. Almost all structures on the surface of the head are sensitive to pain. These include the arteries and muscles of the scalp and upper neck, the orbital contents, the lining of the nasal cavities, and the external and middle ears. When these intracranial and extracranial pain-sensitive structures are disturbed, headache develops.

There are basically four mechanisms by which these structures can be irritated to result in headache:

1. *Dilation of the cranial arteries.* Distention of these vessels results in vascular headaches, which include the head pain associated with such conditions as seizures, trauma, spinal puncture, hypertension, and migraine. The intensity of such headaches is augmented by procedures that increase vascular distention, such as sudden straining.

2. *Traction upon or displacement of pain-sensitive structures.* Such disturbances cause types of headaches that are seen in brain tumors and other intracranial masses, such as a brain abscess and a subdural hematoma. They are accentuated by sudden or vigorous head movement. When due to sustained displacement of structures anchoring the brain, they may be aggravated by coughing or straining.

3. *Inflammation of cranial structures.* Any process that irritates the pain-sensitive structures can result in head pain. This is characteristically seen in cases of meningitis. However, a similar type of headache mechanism occurs in cases of subarachnoid bleeding and following a pneumoencephalogram.

4. *Muscular contraction.* The constant strain or pull of the muscles at the posterior base of the skull often results in headache. Contraction of other head muscles, such as the frontalis and temporalis, may also contribute to the pain, which is aggravated by moving the head either slowly or rapidly and is unaffected by coughing, sneezing, or vascular changes.

HISTORY OF HEADACHE

A careful history often offers some lead as to the possible nature of the headache. In eliciting such a history, a number of specific items should be carefully considered. However, it must always be kept in mind that the description of the pain is in itself often of no great diagnostic value and that there is great overlap in the characteristics of pain of different etiology. In spite of this

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deficiency, a careful history of the headache is frequently helpful and should be obtained in each case.

Age of onset. Since different types of headaches occur in the different age groups, the exact age of onset can be of value. In headaches of long duration, this information may be difficult to obtain.

Location. The location of the headache may be of great importance. One should determine whether the pain is focal or generalized, unilateral or bilateral, orbital or supraorbital, or nuchal. Headaches of eye, sinus, or scalp origin are often well localized to the area of stimulation. Migraine is usually unilateral and involves the head or face. Posterior fossa lesions are often localized to the occipital region, while headaches secondary to neck strain or neck pathology remain localized at their onset to the nuchal region.

Character of pain. Patients use a wide variety of terms interchangeably to describe a headache, thus making this description of the pain of little value in diagnosis. However, since certain types of headaches do produce a characteristic type of pain, it is of value to inquire carefully into the nature of the pain to determine whether it is dull or sharp, pulsating or throbbing, steady or intermittent, aggravated by coughing or sneezing, or accentuated by changes in position or ingestion of alcohol. For example, headaches associated with tumors, hypertension, migraine, and meningitis are pulsating or throbbing in nature; in addition, those due to tumors or hypertension are usually intermittent. On the other hand, headaches due to tension, muscle spasm, and sinus irritation are steady, constant, and persistent. Characteristically, those headaches resulting from displacement of pain-sensitive structures are aggravated by coughing, sneezing, or head movement. It must be kept in mind that the severity of the head pain is not always a measure of the seriousness of the illness. Psychogenic headaches may be very intense, while certain brain tumors may produce only a minimum of distress.

Duration and periodicity. In many cases, the actual course of the headache may be most informative in regard to the seriousness of the illness. In this respect, it is of value to determine the actual duration of each attack, whether it persists for days, weeks, or months. Also, one should determine whether the headache is present both day and night and its relationship to periods of activity and rest, such as week-end headaches. Relation to the menses as well as to

the season of the year may prove helpful. Obviously, a dull headache which has not changed for years would have less significance than one of a few months' duration that had been increasing in intensity. Allergic headaches frequently have a seasonal occurrence, while migraine often appears shortly before or during the menses.

Precipitants. It is often instructive to attempt to elicit any precipitating factors to the headaches. One should inquire into the possible influence of fatigue, tension, heavy work, allergy, noise, eye strain, sinusitis, drugs, alcohol, and injections.

Associated findings. Often, one must depend upon the associated signs to help arrive at a diagnosis. Visual disturbances occur in migraine, a brain tumor, or an aneurysm. In migraine, they often precede the headache and are of a characteristic nature. Focal neurologic findings may suggest the presence of an aneurysm or a brain tumor. Tightness and soreness of the neck musculature can be found in muscular contraction headaches.

In every patient in whom the headache may be of psychogenic origin, a careful history should be obtained regarding possible family or environmental factors that may be playing a role in its production or aggravation.

COMMON HEADACHE TYPES

Ocular headaches. These are caused by errors of refraction, disturbances in ocular muscle equilibrium, inflammation of intra- and extraocular structures, and increased intraocular pressure. They are most often seen in individuals who use their eyes a great deal, such as pianists, typists, bookkeepers, and students.

These headaches are usually bilateral and circumocular. They are related to visual effort and therefore increase in severity as the eyes are used. They are absent on awakening and generally appear in the afternoon when they gradually increase in intensity. Often, during week ends when the eyes are rested, these headaches are minimal or fail to appear.

Sinus headaches. The location of these headaches depends upon the sinuses involved. In frontal sinusitis, the pain occurs over the frontal region; in maxillary sinusitis, it is felt over the cheek and the upper teeth; and, in ethmoiditis, it occurs behind the eye or the temporal region.

The pain is of a deep, dull, nonpulsating quality. It is most severe on arising and is increased by stooping, exertion, coughing, or sneezing. Engorgement of the sinus mucosa, such as occurs with excitement, alcohol ingestion, head-down positions, or menstruation also aggravates

these headaches. Emotional stress can accentuate the pain by engorgement of the nasal mucosa.

Treatment depends upon the nature of the sinus involvement. Vasoconstrictors of the nasal mucosa usually offer immediate relief and can be used as a diagnostic procedure.

Brain tumor headaches. These headaches are not due to increased intracranial pressure but to traction on pain sensitive structures because of displacement of the brain or internal hydrocephalus. Neither the character nor the location of the pain is dependable in the diagnosis or localization of the lesion.

The headaches are mild and intermittent at the onset and last only a few minutes. Later, they increase in intensity and become severe, deep, steady, and, at times, agonizing. They do not interfere with sleep but often awaken the patient at night. The pain is increased by stooping, straining, coughing, or sudden jarring of the head. Terminally, the headaches may become constant, generalized, and very intense.

The diagnosis is established not by the character of the pain but by the associated findings. The presence of focal signs, such as diplopia, reflex changes, progressive mental alterations, or papilledema promptly indicates the diagnosis.

Treatment consists of surgical intervention.

Headaches with other intracranial masses. Other intracranial masses cause headaches by the same mechanism as brain tumors.

Subdural hematoma. This lesion is associated with steady, dull, unremitting headache which is often unilateral at its onset. The associated history of head injury and the fluctuating state of consciousness generally suggest the diagnosis.

Aneurysms and angiomas. These result in periodic, unilateral, throbbing headaches which are often retroorbital. The diagnosis is suggested by the associated cranial nerve findings or episodes of stiff neck and subarachnoid bleeding.

Inflammation headaches. Head pain is a constant finding in acute bacterial meningitis and is felt to be due to some chemical substance that stimulates the pain nerve endings. The headache is a throbbing, persistent pain often localized to the occipital region. It is aggravated by head movement and jugular compression and relieved by rest and quiet. In chronic meningitis, the pain is less intense but often worse at night. The diagnosis is made by the associated nuchal rigidity and spinal fluid pleocytosis. Treatment consists of adequate therapy for the causal inflammation.

Pneumoencephalography. This procedure is usually followed by intense generalized headache which is accentuated by head movement.

The head pain is accompanied by sweating, pallor, and nausea. These patients should be kept in bed, and fluids should be forced. Sedation, analgesics, oxygen inhalation, and intramuscular surgical pituitrin have all been advocated as being helpful.

Subarachnoid hemorrhage headaches. The most common cause of such bleeding is a ruptured congenital aneurysm. The initial headache is produced by traction and displacement of pain-sensitive blood vessels, while the more chronic delayed headache results from secondary reactions of the blood vessels and meninges.

The initial headache is almost pathognomonic. It is a sudden, very intense, throbbing suboccipital pain which may radiate to the neck and is often associated with vertigo, neck rigidity, and drowsiness. The presence of blood in the spinal fluid substantiates the diagnosis. If the patient survives the acute episode, a dull diffuse headache may persist for weeks or months.

Vascular distention headaches. There are a large number of headaches that are due to dilation of some part of the cranial arterial tree. These headaches are all improved by procedures that lower the cranial arterial pressure.

Hangover headache. This is a familiar headache resulting from the vasodilation produced by the overindulgence in ethyl alcohol. It is a diffuse, throbbing pain aggravated by head movement and reduced by carotid pressure.

Caffeine withdrawal headache. This type is usually seen when caffeine is suddenly withheld after excessive administration or use. It is a deep, generalized, occipital pain aggravated by coughing and sneezing and often associated with lethargy and rhinorrhea. It can readily be terminated by the administration of caffeine or Benzedrine.

Postseizure headache. A moderately intense, generalized head pain lasting for hours and accompanied by lethargy develops in many individuals after a convulsion.

Posttraumatic headache. In about 50 per cent of individuals, headache develops following an injury. This type of headache often has no characteristic feature. It may be recurrent, periodic, or throbbing and may be localized or diffuse. The head pain is often accompanied by vertigo and both are accentuated by postural change, emotional stress, or physical fatigue. In many individuals, the headache persists for years as a dull, diffuse, steady discomfort that may be associated with memory impairment.

A number of drugs have been used with some benefit, such as niacin, papaverine, Ronicol, and various analgesics.

Postspinal headache. In certain individuals, intense headache may follow a spinal puncture. It has been suggested that this headache is due to dilation of and traction upon various pain-sensitive structures due to prolonged leakage of spinal fluid through the puncture hole. Characteristically, the headache occurs when the head is raised and immediately disappears on lying down. It is apparently uninfluenced by drugs.

An attempt should be made to prevent these headaches by the use of very fine spinal puncture needles. If headaches do occur, hydration and immediate recumbency are suggested.

Hypertension headache. This is often a major complaint in hypertension. It is a very painful, throbbing pain localized either to the frontal or occipital areas. It occurs on awakening and is often relieved by sitting up or moving about. The frequency and severity are not related to the elevation of blood pressure.

These headaches are not relieved by hypotensive drugs. They are improved by ergotamine tartrate but respond best to analgesics.

Migraine. The exact cause of migraine is unknown. The headaches are produced by dilation of branches of the external carotid artery. There is a strong familial tendency to these headaches, with many members of a family suffering from the same type of disturbance. The role of allergy, toxins, or the endocrines has not been established, although the occurrence of these headaches around the sinuses suggests some associated endocrine factors. Psychologic factors no doubt are extremely important as a precipitating mechanism. These patients invariably are intense, driving, perfectionistic, compulsive individuals who constantly maintain a high degree of internal tension.

Migraine often begins during the second decade of life and implicates all social and economic groups, being most common among members of professional groups. The attacks vary greatly in frequency and intensity even in the same individual.

The clinical features are most variable. Many patients have prodromal symptoms which may precede the headache by days, thus furnishing a warning of the impending headache. These prodromes may consist of a feeling of euphoria or well-being, an increased appetite, an increased mental acuity, an increased sex drive, or a feeling of depression or lassitude.

Many migraine headaches are immediately preceded by an aura due to a transient vascular constriction. Visual aura are the most common and consist of scintillating scotoma, seeing flashing lights; visual field defects, central hemianop-

tie scotoma or complete hemianopsia; or teichopsias, vague visual disturbances, such as a feeling of water over the cornea, snow falling, or cobwebs before the eyes. Other aura may predominate, such as transient episodes of hemihypesthesia, paresthesia, aphasia, ophthalmoplegia (diplopia, ptosis), or gastrointestinal complaints.

The headache usually follows immediately after the aura. Characteristically, it is unilateral at the onset but may become diffuse as it persists. It is an intense, throbbing pain which may be localized to any site—frontal, temporal, or orbital. It often appears in the early hours of the morning and diminishes by evening. It may persist for days but does not seem to interfere with sleep. There is an associated nausea, vomiting, photophobia, and edema of the lids. In severe cases, marked prostration occurs and the patient may become dehydrated because of the vomiting.

Many patients experience the onset of aura without the appearance of the headache. These are called *migraine equivalents* and are named according to the nature of the aura. Because of the absence of the headaches, the migraine equivalents are often overlooked and the patient is investigated for some somatic disease. In *abdominal migraine*, the aura consists of periodic abdominal pain, nausea, vomiting, and bloating. *Precordial migraine* presents with chest pain, tightness in the chest, and palpitation. Other forms of migraine equivalents consist of pelvic migraine, ophthalmic migraine, and ophthalmoplegic migraine. In the presence of a suggestive history of migraine equivalent, one should check carefully for the possible history of headache. Usually, on careful questioning, it is elicited that an occasional episode is followed by head pain and the diagnosis then becomes apparent.

Migraine should be treated both psychologically and pharmacologically. Ergotamine products are the most useful in this illness. Many combinations are now available: (1) Cafergot (ergotamine tartrate and caffeine) orally, 2 tablets initially, followed by 1 tablet at one-half hour intervals until relief is obtained up to a total of 6 tablets; (2) Cafergot suppositories—1 every one-half hour up to 3 suppositories; (3) 0.5 mg. of intramuscular ergotamine tartrate or 1 mg. of dihydroergotamine tartrate (D.H.E. No. 45); and (4) Wigraine in the same dosage as Cafergot.

Ergotamine is contraindicated in organic heart disease, hypertension, pregnancy, hepatic disease, and septic states. When used, it must be given at the immediate onset of the headache. After the pain has become severe, the ergotamine

products are not very helpful. At this stage of the headache, the patient should be treated with large doses of analgesics and hypnotics.

Since personality and tension factors often precipitate these headaches, therapy aimed at these factors should comprise a definite part of the treatment program.

Tension headaches. These are by far the most common type of head pain encountered in general practice. They generally do not appear as an isolated complaint but are accompanied by a wide range of symptoms, such as palpitation, bloating, nausea, urinary frequency, diarrhea, generalized fatigue, and cardiac symptoms. Patients with tension headaches are irritable; have frequent crying spells; are unable to sleep; and, on examination, show tremor of the hands, hyperreflexia, and hyperhidrosis.

These headaches are dull, persistent, and diffuse and have a bandlike quality. They may persist for weeks or months with no change in quality, interfering with the patient's sleep and general activity. There is often an associated pain and tightness of the neck muscles.

Medicinal therapy is used to reduce tension and alleviate the pain. This is accomplished by the use of analgesic-sedative combinations, such as Fiorinal, Daprisal, the barbiturates, and some

of the milder tranquilizers. Psychotherapy directed toward relieving emotional stress and aiding in environmental adjustment is the most important part of treatment of this type of headache.

Muscular contraction headaches. Any condition that results in tightness or irritation of the neck musculature may result in a headache which, at its onset, is occipital and gradually spreads to become diffuse. This type of headache occurs with postural strain and is seen in typists, draftsmen, or students. It also occurs in neck trauma secondary to osteoarthritis, whiplash injuries, and other bony changes in the cervical spine. Less commonly, it is seen in cervical cord pathology, such as meningeal and rootlet lesions.

The pain is a dull, constant, diffuse ache that begins in the nuchal region and spreads to the occiput. It persists for months, does not interfere with sleep, and responds to analgesics. It is usually associated with soreness and tightness of the neck muscles.

Treatment is directed toward the neck pathology. Relief is often obtained by placing a board between the patient's mattress and spring and sleeping without a pillow. Trancopal, 600 mg. daily, aids in muscle relaxation. If the pain persists, cervical neck traction may prove beneficial.

EYE TESTS with the same powdered inhalant allergens that are employed in scratch tests are safe, practical, and useful adjuncts to skin tests. Eye tests are especially helpful (1) to determine whether or not patients with positive skin reactions but negative or doubtful clinical histories have clinical sensitivity, (2) as a means of uncovering unsuspected etiologic factors in patients with negative skin reactions and indefinite histories, and (3) to affirm the nonclinical nature of positive skin reactions in patients with negative histories whose reactions to eye tests also are negative.

The powdered allergen is instilled using a small stainless steel wire with a terminal loop 1 mm. in diameter. The loop is dipped into the allergen and brought close to the mucosa of the pulled-down lower eyelid; the wire is then flicked, transferring the allergen in the conjunctival sac.

Tests with all the important inhalant allergens can be completed in a short time, using both eyes, and done simultaneously with or independently of skin tests.

LOUIS TULL, L. N. EFFEESON, K. GUPTA, and CHARLES KRUEGER: Eye tests with inhalant allergens. *J. Allergy* 30:492-505, 1959.

Head Injuries

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THE MOTOR VEHICLE is probably responsible for more head injuries than any other single agent. These injuries may range in severity from trivial to fatal. They may involve the patient's scalp, skull, meninges, intracranial blood vessels, and brain in varying proportions, so that an infinite variety of clinical pictures and problems is presented. The present discussion will be limited to but three aspects of this general problem.

CARE OF THE SEVERELY INJURED PATIENT

When a patient progressively regains consciousness following a head injury and when his vital signs are only temporarily upset, it is apparent that his injury is mild or moderate in character. Such an individual almost inevitably recovers no matter what sort of treatment is afforded. He must, of course, be observed to detect any reversal in his clinical course.

The severely injured patient, on the other hand, remains more or less deeply unconscious and, at times, restless for a prolonged period. There frequently is external evidence of trauma about the head accompanied by bleeding from the nose, mouth, and ears. The patient's vital signs are usually considerably upset with rapid, shallow, and noisy respirations, a high pulse rate, and fluctuating blood pressure. With time, the temperature tends to rise. The patient frequently exhibits either focal or general neurologic abnormalities. It is in patients of this character that treatment may be lifesaving if pursued with sufficient energy.

If the patient presents evidence of shock, this must be combated immediately and in the usual fashion. Significant degrees of shock in a patient with a head injury should alert one to the possibility of associated injuries. These injuries may be difficult to detect in the unconscious patient. Particular attention should be given to fractures

of the spine, especially the neck; chest wounds; and abdominal injuries with possible ruptured viscus.

The most important single consideration in the nonsurgical care of these patients is the provision of an adequate oxygen supply. As is well known, the brain is peculiarly dependent upon a continuing oxygen supply, and anoxia of the nerve cells for only a few minutes may suffice to produce irreparable damage. The respiratory exchange of the patient with a head injury is frequently hampered by the rapid, shallow character of his breathing and by the accumulation of secretions in the tracheobronchial tree as well as by the tendency of his jaw and tongue to fall backward, producing mechanical obstruction. The simplest means of overcoming these problems is to place the patient in the coma position, that is, on his side with the face turned toward the bed, allowing the jaw and tongue to fall forward and permitting secretions to run out of the mouth. An airway may be inserted, and the throat and the larynx may be cleared by suction.

Whenever there is any serious degree of respiratory difficulty, tracheotomy should be carried out immediately. This measure probably saves more lives among this group of patients than any other single maneuver.

Oxygen should be administered to the patient, preferably by means of a catheter inserted through the nose into the throat or through the tracheotomy tube if one is in place. An oxygen tent should not be employed, since it is a less efficient means of increasing the oxygen content of the alveolar air and tends to isolate the patient from the nurse.

A rising temperature is a characteristic result of virtually every serious brain injury. If this progresses to extreme levels, it is in itself fatal. Also, as the temperature rises, the oxygen consumption of the brain is increased, and a state of relative anoxia is thereby more easily produced. This hyperthermia is neurogenic in origin and must not be regarded as evidence of infection. If the patient's temperature rises above 103° F., it must be actively reduced by strenuous nursing measures, including the removal of bed

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clothing, alcohol sponges, and the application of ice packs to the body. If a hypothermic blanket is available, it may be employed.

Deliberate hypothermia by means of the hypothermic blanket, reducing the patient's temperature to the lower 90 or upper 80° range, may be lifesaving for some of these patients. This hypothermic state may be maintained for several days, during which time much of the swelling of the brain will recede. Care must be taken not to lower the patient's temperature to a range in which cardiac arrhythmias may occur.

It has long been recognized that the intravenous injection of hypertonic glucose or sucrose solutions is of no value during the management of cerebral edema resulting from head injury. Recently, however, a 30 per cent solution of urea has become available, which, when given intravenously, has a marked ability to shrink the brain. A problem which immediately arises is that of deciding when the patient is suffering from cerebral edema. Practically, this cannot be determined from clinical findings and is best established by visualization of the tight brain through a burr hole.

After the first day, patients with head injuries should be provided with a normal fluid intake, and their electrolyte balance must be maintained in the usual fashion. If unconsciousness persists more than a few days, parenteral feedings should be instituted. Restlessness, irritability, and convulsive twitchings or seizures may present a serious problem. It is important never to employ sedatives which depress the respiratory function. The chief offender of this type is morphine, and this drug should never be used in the management of these patients. Paraldehyde, chloral hydrate, and drugs of the phenothiazine group may be used for the control of restlessness and phenobarbital and Dilantin for the control of convulsions. A severe head injury is an extremely stressful situation. It is probably wise to place such patients on a drug of the cortisone type in adequate dosage for a few days and then gradually taper it off as the patient improves. This is particularly important if the patient has previously received steroid medication.

A lumbar puncture is of little use in the acute stage of severely injured patients. The spinal fluid is almost always found to be bloody. If the intracranial pressure is high, or if a large clot is present displacing the brain, a lumbar puncture may be dangerous. In any event, if lumbar puncture is done, the Queckenstedt should never be carried out in a patient with a head injury.

Similarly, early procurement of routine roent-

genograms of the skull is rarely beneficial, although it does have a reassuring effect upon the patient's relatives. Portable roentgenograms of the skull may serve to exclude the presence of a depressed skull fracture or a fracture in the temporal area. They should, of course, be supplemented by regular studies at a later date.

Carotid angiography is a measure of considerable usefulness in the management of these patients when there is reason to suspect an intracranial accumulation of blood or fluid.

The electroencephalogram has little place in the management of the acute phase of these injuries but may be useful in the later stages of management.

OPERATIVE INDICATIONS

Scalp lacerations usually require suture, but this is not an emergency and may well be deferred if the patient's general condition is precarious. One should always explore a scalp laceration with the tip of a gloved finger to detect an underlying depressed skull fracture.

Depressed skull fractures rarely constitute a surgical emergency. In many of these patients, the associated brain injury is relatively mild or moderate, and the patient is in no danger. Such individuals may readily be transported to another hospital if this move will facilitate the care of their depressed fracture. A sterile dressing should be applied to the wound, and the patient should be placed on antibiotic medication. It is well not to tamper with the wound beyond establishing the presence of a depressed skull fracture. In the definitive surgical management of these cases, the first consideration is to determine whether the underlying dura has been torn and the brain lacerated. If it has, careful debridement of the injured brain must be carried out, and the dura must be carefully repaired. This minimizes the amount of cerebral scarring and hopefully reduces the incidence of convulsive seizures. As for the depressed bone fragments themselves, all of the larger pieces should be conserved and replaced in their normal position unless they are obviously contaminated, in which case they must be discarded. In most instances, it is possible to elevate the depressed fracture and restore the continuity of the skull without leaving any significant skull defect. However, should it be necessary to remove bone, the resulting defect may be closed at a subsequent time, using either a metallic or plastic plate. The initial wound is allowed to heal thoroughly before any foreign materials are introduced.

The only real neurosurgical emergency pre-

sented by the patient with a head injury is that occasioned by the development of an expanding intracranial clot. This usually results from the tearing of the middle meningeal artery by a fracture which crosses the artery in the temporal region. Therefore, any head-injured patient who presents x-ray evidence of a fracture line crossing the middle meningeal groove of the temporal bone should be watched carefully for a number of hours. The classical example of a patient with a middle meningeal hemorrhage is one who suffers a blow to the head which produces transient unconsciousness and then recovers and seems quite uninjured. After this so-called lucid interval, headache, progressive coma, and hemiparesis develop, the pupil on the side of the hematoma becomes dilated. Typically, the patient's blood pressure rises while his respiratory and pulse rates fall, sometimes to surprisingly low levels. In a typical example, diagnosis is readily made. Surgical removal of the accumulating clot produces a dramatic cure if carried out before irreparable damage is done. Search for such a clot should be made whenever reasonable suspicion of its presence exists, since, unfortunately, only a minority of patients with an extradural clot present the typical clinical syndrome previously described.

With the increasing availability of neurosurgical facilities, the tendency to carry out exploratory trephination in patients with head injuries has increased. Indications for such intervention are somewhat vague and variable. In general, the patient who remains unconscious or whose comatose state appears to be deepening and who otherwise fails to improve under adequate management is regarded as a candidate for surgery, particularly if he presents some focal findings, such as increasing unilateral weakness or paralysis. It should be pointed out that surgery can be helpful only if a space-occupying lesion of some sort is found and evacuated, that is, a blood clot or fluid accumulation of sufficient volume to compress the brain. If the patient's principal problem is injury to the brain substance itself, surgery will not benefit him. One does not "decompress" the brain by placing a few small holes in the skull.

The technic of exploratory trephination varies among surgeons. Commonly, the patient's head is shaved, placed in a head rest, and draped in such a fashion as to expose the entire calvaria.

It is then possible to place multiple burr holes in the frontal, temporal, and parietal regions on each side as may seem indicated. Others, including the author, prefer in most instances to place a single 1½- to 2-in. trephine craniotomy just above the ear. In either case, any fluid collections encountered are evacuated. The brain may be cannulated as well, searching for intracerebral clots.

PSYCHOLOGIC MANAGEMENT

Injuries to the head differ from all other types of injuries in that they are directed toward that part of the body which, in effect, contains the patient's self and personality. Everyone knows that injuries to the head frequently cause death or serious aftereffects. For this reason, it is perfectly reasonable for the person who has received a head injury, even a minor one, to be struck by the conclusion that "I might have been killed." This is very frightening to the patient and may have far-reaching consequences in the development of symptoms arising after a head injury.

In the management of patients with an injured head, particularly those with mild and moderate injuries, the physician must be very careful not to add to their already existing anxieties. To this end, he should be careful in his choice of words in discussing the patient's injuries with him or his relatives. Terms like "skull fracture" and "concussion" may carry a much more serious import to the patient than to the physician. The patient should be reassured regarding the mild or minor character of his injuries if, in the physician's opinion, this is the case. Early ambulation is desirable. The patient should be allowed to be up just as soon as his subjective symptoms of headache, dizziness, and fatigability permit. The administration of a tranquilizing drug for a period of time may be of considerable benefit.

Since many head injuries arise in situations in which there is a question of liability and damages, the physician frequently finds himself in a position of seeming conflict with the patient's legal advisor whose concern to delineate the full extent of the patient's real and potential injuries may unwittingly serve to increase and prolong his subjective symptoms. In such an instance, the physician must decide whether he is the patient's doctor or his lawyer.

Aids in Investigation of the Cyanotic Patient

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CYANOSIS is a term derived from the Greek "kyanos," meaning dark-blue. It was defined medically by Lundsgaard and Van Slyke¹ as blueness of the skin, mucous membranes, or organs caused by changes in capillary blood—usually, unusual amounts of reduced hemoglobin. They listed several modifying factors which might interfere with the visual impression of true cyanosis. These included (1) the thickness, color, and opacity of the tissue overlying the capillaries; (2) the number and length of the vessels and their state of dilation or constriction; and (3) variations in plasma color caused by dyes or drugs.

Comroe and Botelho² conducted a well-controlled study which demonstrated the unreliability of cyanosis as a sign of arterial hypoxemia. The ability of trained and practiced observers to detect cyanosis was evaluated by comparing color estimations with arterial oxygen saturations as monitored by an oximeter. It was revealed that the majority of 127 observers were unable to detect definite cyanosis until the arterial oxygen saturation fell to approximately 80 per cent. Twenty-five per cent of the observers failed to detect cyanosis until the oxygen saturation was 71 to 75 per cent. This reaffirms the classic finding of Lundsgaard³ that the clinical appearance of cyanosis is dependent on the absolute amount of reduced hemoglobin present and that this value is between 4 and 6 gm. per 100 cc. of blood, usually 5 gm. per cent.

For the sake of discussion, we have chosen to consider only cases of so-called "central" cyanosis as distinguished from "peripheral" cyanosis. Peripheral cyanosis is the discoloration of the skin caused by excessive amounts of reduced hemoglobin in the capillaries, which are, however, due to local factors. These factors result in reduced rate of flow of blood through the affected part. This leads to abnormal utilization of

oxygen which is delivered to the tissues in normal amounts in arterial blood. The conditions leading to peripheral cyanosis are: for example, acrocyanosis, congestive heart failure in which arterial hypoxemia is rarely present except in cases of severe pulmonary edema,⁴ venous stasis, exposure to extremely low temperatures, and shock. These cases can usually be differentiated by means of proper physical examination and history.

French and Douthwaite's *Index of Differential Diagnosis*⁵ refers to 40 or more distinct disease entities which may be the cause of cyanosis. All of these diseases can be conveniently classified as follows (modified after Selzer⁴):

1. Abnormalities of hemoglobin
2. Pulmonary causes
 - a. Hypoventilation (alveolar)
 - b. Alveolar ventilation-perfusion imbalance
 - c. Oxygen diffusion defect
3. Cardiovascular anomalies
 - a. Pulmonary arteriovenous fistula
 - b. Congenital cardiac anomalies leading to right-to-left shunts

One or more of these mechanisms is operative in any patient whose arterial blood is sufficiently hypoxemic to produce the appearance of cyanosis. In addition, a large number of individuals have no visual evidence of cyanosis and yet are significantly hypoxemic, since, as has been noted previously, a fairly severe degree of hypoxemia must be present before cyanosis is detectable. The various factors producing arterial hypoxemia and/or cyanosis can be quite definitely differentiated one from another by the use of certain laboratory studies as will be shown.

ABNORMALITIES OF HEMOGLOBIN

The normal "s" shape of the oxygen-hemoglobin dissociation curve is familiar. The normal oxygen carrying capacity of hemoglobin may be reduced by several sets of circumstances, including (1) a decrease in pH, which causes a "shift to right" of the entire curve; (2) changes in the electrolyte

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content of the erythrocyte, about which very little is known; and (3) by changes in the hemoglobin molecule itself, which completely destroy its oxygen carrying properties. Of these factors, the first and second are of relatively little quantitative importance, since changes profound enough to seriously impair the oxygen-hemoglobin system, that is, to produce significant hypoxemia, are probably not compatible with life. The third set of circumstances would include cyanhemoglobin, carboxyhemoglobin (carbon monoxide-hemoglobin complex), methemoglobin, and sulfhemoglobin. Cyanhemoglobin and carboxyhemoglobin, although incapable of binding oxygen, are not causes of cyanosis, since they are bright red pigments. Methemoglobin and sulfhemoglobin, on the other hand, in addition to being incapable of carrying oxygen, are bluish pigments. The relative capacity of methemoglobin and sulfhemoglobin to produce cyanosis is indicated by the fact that, whereas 5 gm. per cent of reduced hemoglobin is necessary to produce cyanosis, 1.5 gm. per cent methemoglobin and less than 0.5 gm. per cent sulfhemoglobin are sufficient to produce recognizable cyanosis.⁶

Normal hemoglobin contains iron in the ferrous form, and the valence of the iron does not change with oxygenation and deoxygenation. Methemoglobin contains iron which has been oxidized to the ferric form. This is an easily reversible change, and the normal erythrocyte contains no more than 1 to 2 per cent methemoglobin. In plasma, on the other hand, methemoglobin is not as efficiently reduced, and free plasma hemoglobin comes to equilibrium with only slightly greater than 50 per cent of the hemoglobin in the ferrous form.

Finch⁶ collected from the literature 16 cases which he thought demonstrated primary methemoglobinemia. These patients were thought to have defects in the normal catalytic and enzymic processes necessary to reduce methemoglobin. Such patients generally have about 40 per cent methemoglobin. They have, in addition, low ascorbic acid levels in the serum. Treatment with ascorbic acid in daily, oral doses of 100 to 500 mg. reduces the methemoglobin content to 8 to 10 per cent of the total hemoglobin.

Secondary methemoglobinemia is due to drugs or to enterogenous methemoglobinemia, which is apparently caused by absorption of nitrites from the bowel in such large amounts that ferrous hemoglobin iron is oxidized to ferric iron in greater amounts than can be reconverted by the normal body mechanisms. Drugs which can be responsible for methemoglobinemia are:^{6,7}

AROMATIC DRUGS

Aniline	Alphanaphthylamine
Anilinoethanol	Paraminopropiophenone
Phenacetin	Phenylhydroxylamine
Acetanilid	Tolylhydroxylamine
Methylanacetanilid	Nitrobenzene
Hydroxylacetanilid	Dinitrobenzene
Prontosil	Trinitrotoluene
Sulfanilamide	Nitrosobenzene
Sulfapyridine	Paranitraniline
Sulfathiazole	Primaquine
Phenylenediamine	Pamaquine
Aminophenol	Sulfones and ethylsulfones
Toluenediamine	

ALIPHATIC AND INORGANIC DRUGS

Sodium nitrite	Amyl nitrite
Hydroxylamine	Ethyl nitrite
Dimethylamine	Bismuth subnitrate
Nitroglycerin	Ammonium nitrite

Sulfhemoglobin is another abnormal pigment which is never present in the body under ordinary circumstances. The exact nature of sulfhemoglobin is not known, but one possibility is that one of the nitrogens in the pyrrole ring is displaced by sulfur. The drugs which cause methemoglobinemia also cause sulfhemoglobinemia. Sulfhemoglobin cannot be converted to normal hemoglobin but must be metabolized and excreted.

Methemoglobin and sulfhemoglobin may be detected in several ways. French suggests the following approach. When an abnormal hemoglobin pigment is suspected, blood should be drawn, anticoagulated, and centrifuged. If the plasma is clear, hemolysis and abnormal serum pigments are excluded as a cause of cyanosis. An attempt should then be made to oxygenate the blood by shaking it in the air or bubbling air or oxygen through it. If the blood remains dark, abnormal intracellular pigments are present. The blood should then be diluted 10 to 100 times with water and examined with a hand spectroscope. For methemoglobin, a dark band appears at 630 μ ; for sulfhemoglobin, at 618 μ . The band for methemoglobin disappears immediately when 2 to 3 drops of 5 per cent potassium cyanide are added, whereas the band for sulfhemoglobin remains fixed. The band for sulfhemoglobin is dispersed by the addition of hydrogen peroxide to the solution.

The abnormal pigments can be shown also by plotting their absorption spectra with the Photometer and demonstrating increased absorption at the proper wave lengths. The pigments can be quantitatively estimated by colorimetric methods, and gasometric methods can be used to determine the amount of normal hemoglobin capable of carrying oxygen and this value compared with the total hemoglobin content.

These causes are numerically the most important in the adult patient. It is important that the cause of hypoxemia be determined accurately, since the consequences of prolonged oxygen deficiency are profound and because many of the underlying conditions can be corrected.

Hypoventilation. This simply means decrease in the amount of oxygen which reaches the areas of the lung, principally the alveoli, at which gaseous exchange between blood and environmental atmosphere occurs. It should be clear that alveolar ventilation is the critical value. A patient may appear to the eye to be adequately ventilating and yet, in truth, hypoventilation may be taking place in his alveolar spaces.

Alveolar ventilation per minute = (tidal volume - dead space) \times respiratory frequency or $V_A = (V_T - D) \times f$. (See addendum for glossary of symbols and terms used in pulmonary physiology). It is possible to measure dead space in the physiology laboratory, but the methods devised are rather complex and not suitable to be used as a routine test. However, tables are available for normal dead spaces in various groups of individuals and disease states, and, since little variation is encountered when dealing with anatomic dead space, these values may be used without significant error.⁸ The so-called "pickwickian" syndrome of alveolar hypoventilation, which is associated with obesity alone, is the most dramatic example of this type of difficulty. Alveolar ventilatory deficits may also occur in patients with central nervous system lesions and with neuromuscular disorders.

Of far greater importance as far as adequate ventilation is concerned is the physiologic dead space, since this volume is dependent on relationships between ventilation of alveoli and pulmonary capillary blood flow. In most cases in which pulmonary disease is significant, ventilation is uneven in relation to blood flow. In these instances, the anatomic and physiologic dead space volumes are widely divergent. For the sake of discussion, this problem will be considered separately, as indicated in the outline previously noted.

Alveolar ventilation-perfusion imbalance. It is clear that gaseous exchange between inspired gas and pulmonary capillary blood depends on a close anatomic relationship between the two media and on the barrier between the two being of such a nature that diffusion of the two important respiratory gases, oxygen and carbon dioxide, is relatively unimpeded.

If pulmonary blood is not brought into contact

with a diffusing surface where such exchange can take place, it follows that this blood will be returned to the systemic arterial circulation with carbon dioxide and oxygen identical with that of venous blood. This process may be thought of as constituting a physiologic, as contrasted with an anatomic, venous-to-arterial shunt. Riley and Cournand⁹ devised a truly ingenious method of analyzing these ventilation-perfusion relationships, utilizing rather complex concepts involving "ideal" alveolar air. Suffice it to say at this point that the physiologic shunt can be differentiated from both diffusion defects and from anatomic venous-to-arterial shunts by relatively simple procedures, as will be shown.

Qualitatively, uneven ventilation of the lungs can be detected in a simple manner in the laboratory. Grossly uneven ventilation may be revealed by having the patient breathe pure oxygen for seven minutes. At the end of this period, a sample of alveolar air is collected—the last few milliliters of a forced expiration—and analyzed for nitrogen. A value greater than 1.5 per cent N₂ remaining in the lungs is indicative of uneven ventilation.¹⁰ More subtle uneven pulmonary mixing may be detected by continuously monitoring the expired air for nitrogen with the nitrogen meter while the subject breathes pure oxygen,¹¹ and preparing a nitrogen washout curve. The values on the normal curve are known. Even minor degrees of uneven pulmonary mixing may be detected by comparing the patient's curve with the normal.

Physiologic dead space can be measured indirectly by the method of Riley and Cournand⁹ by substituting in the following formula:

$$V_D = \left(\frac{F_{ACO_2} - F_{ECO_2}}{F_{ACO_2}} \right) V_E$$

(F_{ACO_2} is assumed to be equal to P_aCO_2)

Likewise, the fraction of total cardiac output flowing through the total venous-to-arterial shunt (physiologic and anatomic shunt) may be calculated by substituting in the formula:

$$\frac{\dot{Q}_s}{Q} = \frac{C_{aO_2} - C_{cO_2}}{\bar{C}_{vO_2} - C_{cO_2}}$$

End pulmonary capillary blood oxygen content (C_{cO_2}) must be derived by Riley and Cournand's method, since it is not measurable. Oxygen content of arterial blood and mixed venous blood are measurable. If Q , cardiac output, is determined, the actual flow through the physiologic

shunt may be calculated. The normal physiologic shunt is approximately 6 to 8 per cent of total cardiac output.¹²

Uneven ventilation, in general, is due to regional changes in elasticity, obstruction, check valves, and disturbances in expansion. Uneven blood flow may be due to anatomic shunts; embolization or occlusion of pulmonary artery branches; regional reduction in the pulmonary vasculature, as occurs in emphysema and fibrosis; and regional alterations in blood flow, as may be caused by pneumothorax, compression by tumors, pulmonary congestion, or pneumonia.

Oxygen diffusion defect. An abnormal barrier to the passage of oxygen from the alveoli into the pulmonary capillary blood constitutes a diffusion defect. Some disease processes which may cause diffusion defects—so-called alveolar-capillary block—are sarcoidosis, pulmonary berylliosis, scleroderma, lymphangitic metastatic carcinoma, and pulmonary edema.

Diffusing capacities of the lung can be measured quantitatively by the use of either carbon monoxide¹³ or oxygen¹⁴ as the test gas. For the most part, these determinations are too complex and laborious to be used as routine laboratory procedures. Also, the quantitation of a diffusion defect serves no useful clinical purpose. As will be shown, the presence of a diffusion defect can be qualitatively demonstrated with relative ease.

CARDIOVASCULAR ANOMALIES

This classification includes only the true, anatomic, venous-to-arterial shunts:

Pulmonary arteriovenous fistula. The pulmonary vascular bed is unique in that the arterial side of the circulation, wherein higher pressures exist than in the venous side, contains venous blood. Thus, anatomic disturbances, such as hemangiomatosis or acquired arteriovenous anastomoses, result in a right-to-left shunt. Peripheral or systemic circulation arteriovenous anastomoses must result in flow of blood from the arterial to the venous side. Since no admixture of venous blood on the arterial side occurs, cyanosis cannot result.

Congenital cardiac anomalies resulting in venous-to-arterial shunts. In general, it may be stated that history of cyanosis from birth or shortly thereafter and physical and/or roentgenographic findings are usually sufficient to establish diagnosis of a congenital cardiac anomaly as the cause of cyanosis. It may be, however, considerably more difficult to establish the precise anatomic variant present. It is beyond that scope of this paper to discuss the means for making this type of precise anatomic diagnosis.

DIFFERENTIAL DIAGNOSIS¹⁵

Cyanosis caused by diffusion defect, alveolar ventilation-perfusion imbalance, and anatomic shunt may be differentiated by relatively simple, practical measures, which remain to be discussed. Cyanosis due to abnormalities in hemoglobin and to alveolar hypoventilation has been excluded by the measures already outlined. These other causes may be differentiated by the simple expedient of determining arterial oxygen saturations while the patient breathes three different concentrations of oxygen. By simple calculation, the alveolar PO_2 may be ascertained for any given O_2 content of inspired gas. The alveolar-arterial PO_2 difference may then be calculated. If the $a - APO_2$ difference is known for air ($O_2 = 20.93$ per cent), then an increase of this difference when the patient breathes gas with a low (12 to 14 per cent) O_2 content indicates the presence of diffusion difficulties. If then, the patient is allowed to breathe pure O_2 , failure of the arterial PO_2 to reach equilibrium with alveolar PO_2 (theoretic maximum is 673 mm./Hg BTPS) indicates an anatomic shunt. The formula previously used for determining per cent physiologic shunt may be applied here to calculate the per cent of cardiac output passing through the anatomic shunt. Arterial PO_2 may be determined within ± 2 mm. Hg.¹⁶ The normal anatomic shunt, that is, the shunt via the bronchial and thebesian veins, does not exceed 2 per cent.

It should be noted that in many disease processes several, if not all, of these causes of arterial hypoxemia may be operating to a greater or lesser degree.

SUMMARY

The cause, or causes, of cyanosis or of arterial hypoxemia even in the absence of cyanosis may often be determined with considerable accuracy by judicious use of physiologic studies. The measures available to make this differentiation possible on a practical level have been outlined and discussed.

ADDENDUM

In 1950, at a meeting of a group of the leading American pulmonary physiologists, a standard set of symbols and abbreviations was agreed upon. Some of these symbols and abbreviations pertaining to pulmonary physiologic data are included here to aid in the interpretation of various formulas presented both here and in the literature.

PRIMARY SYMBOLS
(Large capital letters)

For gas	For blood
V = gas volume	Q = volume of blood
P = gas pressure	E = concentration of gas
F = fractional concentration in dry gas phase	in blood phase
F = respiratory frequency	S = per cent saturation of hemoglobin with O ₂
D = diffusing capacity	

SECONDARY SYMBOLS

For gas (SMALL CAPITAL letters)	For blood (small letters)
i = inspired gas	a = arterial blood
e = expired gas	c = capillary blood
A = alveolar gas	v = venous blood
T = tidal gas	
D = dead space gas	
BTPS = body temperature and pressure saturated with water vapor	

SPECIAL SYMBOLS

- Dash over any symbol indicates a mean value
- Dot above any symbol indicates a time derivative

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ALTHOUGH IRRADIATION causes remission of acne by preventing new lesions, the effect is only temporary.

The object of radiation therapy is protracted suppression of the large sebaceous glands. This can be accomplished safely by giving 75 r a week for ten to twelve weeks. Permanent suppression of the sebaceous glands cannot be obtained by irradiation; even when 1,500 r is given at a single exposure, an amount that invariably produces permanent atrophy and telangiectasia, some sebaceous regeneration is seen.

Remission lasts for two to three months after completion of therapy.

J. S. STRAUSS: Radiation therapy of acne, *Connecticut Med.* 23:654-655, 1959.

Cat Scratch Disease

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IN DIFFERENTIATING among the various lymphadenopathies of childhood and adult life, a large number of diseases have to be considered. The possible diagnoses include tuberculosis, bacterial lymphadenitis, tularemia, lymphogranuloma venereum, infectious mononucleosis, and tumors such as Hodgkin's disease, lymphoma, and lymphosarcoma. When these conditions have been ruled out, and especially if there is a history of cat scratch or bite or even just cat contact, the diagnosis of cat scratch disease should be considered. This disease is not altogether uncommon, and the typical case is easily recognized once a case has been studied.

Cat scratch fever was described by Debré¹ in 1950. He, Foshay,² and others³ had recognized the association of a cat scratch or cat bite with a benign clinical picture similar to, but different from, that of the usual bacterial and malignant lymphadenopathies. However, it was not until Hanger and Rose³ developed a specific test antigen from the pus of suppurative nodes of patients with this clinical syndrome that these diversely scattered cases could be grouped as a disease entity.

The syndrome is usually described as cat scratch fever or cat scratch disease, but it has also been called cat claw fever, cat bite fever, benign inoculation lymphoreticulosis, and non-bacterial regional lymphadenitis. The last is the

form preferred for coding, using the *Standard Nomenclature for Diseases and Operations*.⁴

EPIDEMIOLOGY

About 1,000 cases of cat scratch disease have been recorded in the literature. These have been reported from nearly every country in the world, although most have occurred in Europe and North America. There is no racial immunity, for, in the areas of high incidence, the disease occurs in the various races in proportion to their population ratios. The disease affects male and female alike. One-half or more of the observed cases have been in children.

The disease has been observed in 3 general epidemics,⁵⁻⁷ which have been restricted to the fall and winter months. It is likely that other epidemics of cat scratch disease have occurred and have not been observed. At least in North America, and probably elsewhere, the disease is most common between September and February, with the peak incidence in the months of November and December.⁷

The disease occurs quite commonly in small family epidemics,⁸⁻¹⁴ but only when there is a family cat, usually a kitten. The association with a young and playful kitten has been noted by several writers and is as characteristic of the sporadically occurring cases as it is of those in family epidemics.^{13,14} The overwhelming majority of cases has contact with such a cat,^{8,13} and, in two-thirds of the cases associated with a cat, the disease can be traced to a definite scratch or bite. Other types of inoculating skin injuries are known, including insect bites, rabbit scratches, dog bites, and skin pricks by inanimate objects, such as pins and fishhooks. Some cases of cat scratch fever have no known trauma to the skin or contact with a cat.

With rare exceptions, the cats associated with cases of cat scratch disease are well, and, when

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the cat has not been well, its illness has proved to be a known disease of cats. The role of the cats in the causation of this disease is further complicated by the fact that they do not show hypersensitivity to cat scratch fever antigen. Until the causative agent of cat scratch disease and its reservoir are found, we can say only that the cat is a peculiarly effective inoculator of the disease.

ETIOLOGY

Cat scratch disease is presumed to be the result of a virus infection because its clinical course is that of an infectious disease, because of its typically infectious type of epidemic, and because bacteria, viruses, fungi, and protozoa have not been found in the diseased nodes. Furthermore, certain experimental evidence supports this conclusion. Debré¹³ was able to produce skin granulomas in rodents by pretreating them with cortisone to enhance their susceptibility to infection and then injecting them intradermally with pus from patients with cat scratch disease. Mollaret and associates¹⁵ also used pus from cases of cat scratch disease as their infective material. Working with monkeys, they produced a granulomatous disease involving the inoculation site and its regional lymph nodes. They also transmitted the disease to 1 of 4 laboratory workers inoculated with the pus. Zwissler⁵ produced a granulomatous disease in 4 persons who were skin tested with inadequately prepared skin test antigen made from pus from a case of cat scratch disease. This material had been heated to 58° C. for one hour on only one occasion. He also produced positive skin tests in patients with cat scratch disease, using antigen prepared after the fifth passage of the infectious pus through rat testes and through chick egg yolk sacs. Unfortunately, none of these investigators was able to isolate the causative agent.

It is probable that this virus is one of low grade infectivity or pathogenicity because others have failed to duplicate their results with rabbits, guinea pigs, cats, mice, rats, monkeys, or chick embryo yolk sacs or to find evidence of virus multiplication with tissue culture techniques.

This infectious agent is presumed to be a member of the psittacosis-lymphogranuloma group of viruses because one of the common diseases of cats, feline pneumonia, is caused by a virus of this group, the pathologic picture of the nodes is similar to that in lymphogranuloma venereum, and about one-half of the patients show low complement fixation titers against *Lyngnam*.

Armstrong and associates¹⁶ have recently ques-

tioned this last relationship because they found similar increases in positive titers in old and young control patients and cat scratch fever patients. However, their cat scratch patients as a group had positive titers significantly more frequently than did their control groups. Perhaps the best suggestive evidence comes from New Zealand¹⁷ where the other viruses of this group are exceedingly rare. There, 23% of cat scratch fever patients and 2% of controls had low positive titers to such antigens.

The cytoplasmic inclusion bodies noted by some investigators^{15,18} are often absent and, when present, are similar to those found in several of the bacterial and fungal lymphadenopathies.¹⁹

CLINICAL PICTURE

Inoculation. The patients have a cat at home or have had contact with a cat 83 to 98% of the time. About two-thirds of patients have had a cat scratch or a cat bite. On occasion, a patient has had instead an abrasion, bite, cut, or other puncture of the skin.

Primary lesion. Usually ten to twelve days after the inoculation, but as early as three days or as late as several weeks, the primary lesion develops at the site of the inoculation. It appears either locally or diffusely as a red and inflamed area about the scab, and, as it develops, it becomes reddish-purple, firm, indurated, and papular. The papule may be covered with fine scales or a vesicle, and a pustule or small ulcer may appear at its apex. The duration of the primary lesion is variable but usually one or two weeks. Reviviscence of the primary lesion with the development of the regional lymphadenopathy is an occasional finding that is largely specific for cat scratch disease.

Regional adenopathy. All clinical cases of cat scratch disease have regional lymphadenitis. The adenopathy develops in the nodes that drain the site of inoculation, without intervening lymphangitis. Typically, the first and second sets, but occasionally the second and third sets, are involved. The diseased nodes are the epitrochlear or axillary nodes in about 60% of the cases, the submandibular or cervical nodes in about 25%, and the inguinal or femoral nodes in about 10%. Atypical locations for the enlarged nodes include the areas beneath the pectoralis, trapezius, and sternocleidomastoid muscles, the mediastinum, the intestinal mesentery, and the midline of the neck. A few cases of generalized lymphadenopathy and hepatosplenomegaly have been seen. The adenopathy develops within one to three weeks after the inoculation in two-thirds and

within two months in over nine-tenths of the cases. On occasion, it may not appear for six or seven months. The enlargement of the nodes is typically heralded by local pain, often severe enough to limit the patient's activities. If tender, the nodes may remain so until they begin to involute.

Within two weeks, 10% have regressed to apparently normal size, 35% in four weeks, and 85% in six weeks. Occasionally, active disease may persist in the infected nodes for several months. If fibrosis occurs, the enlargement may persist for several years.

At onset of adenopathy, the nodes are freely movable. However, as the disease progresses, and if suppuration occurs as it does about half the time, the inflammation extends to the surrounding tissues, matting the nodes together and fixing them to the skin and the surrounding tissues. At this stage, the skin shows erythema, induration, and a local increase in temperature. If the nodes are aspirated at this time, variable amounts of pus are obtained, up to 1 or 2 oz.

If the internal increase in pressure in these nodes is not relieved by aspiration, the associated cellular necrosis may permit cutaneous rupture to occur, with a temporarily draining sinus. Such spontaneous evacuation of the necrotic nodes leads to rapid healing of the nodes and sinus with minimal scarring.

Constitutional symptoms. The systemic reaction to cat scratch disease is exceedingly variable. In some cases it is mild, in some cases faint and ephemeral, but in a few cases severe and prolonged. Fever is the most common symptom, occurring in three-fourths of the cases. It is usually of low intensity (100 to 102° F) but in severe cases may reach 104 or 105° F. It lasts from a day or two to a week in most cases but at times may persist for several weeks. The other generalized symptoms are recorded in the order of decreasing frequency: chills or chilly sensations, headache, backache, generalized aches and pains, malaise, lassitude, anorexia, nausea, and abdominal pain. These systemic symptoms and the patient's appearance of illness are generally less than one would expect from a similar bacterial lymphadenopathy.

ATYPICAL FORMS OF CAT SCRATCH DISEASE

Unilateral conjunctivitis with homolateral preauricular lymph node enlargement, the oculoglandular syndrome of Parinaud, is perhaps the most common of these rare forms.^{20,21} The primary lesion occurs in the eye, which becomes red, swollen, and tender, with the formation of

retrotarsal granulations or granulomas. Within a week or two after the eye is involved, the preauricular lymph nodes enlarge and become tender. The regional cervical lymph nodes may also be involved. The conjunctival lesions regress in one to several weeks, but the adenopathy may persist considerably longer.

Various exanthemas occur in about 7% of cases of cat scratch disease.⁸ Most common are erythematous, morbilliform, and maculopapular eruptions and erythema nodosum. Thrombocytopenic purpura²² has also been reported.

Encephalitis, myelitis, radiculitis, and encephalomyelitis have occurred in cases of cat scratch disease.^{23,24} The 13 patients reported ranged from 4 to 48 years of age, with 9 of them between the ages of 4 and 16 years. The illness began three days to five and one-half weeks, usually seven to fourteen days, after onset of adenopathy. The major symptoms and clinical findings in this syndrome, in order of their frequency of occurrence, were fever, convulsions, coma, muscle weakness, pyramidal tract involvement, neck stiffness, and sensory changes. About one-third of the patients recovered in one week, another third in four weeks, and the rest in three to six months. The cerebrospinal fluid showed moderate pleocytosis and slightly elevated protein in one-third of the patients. A generalized, nonspecific dysrhythmia, with high amplitude curves at 3 to 9 per second, was usually found on the electroencephalogram. All of the patients observed made good recoveries.

The mesenteric lymph nodes may be involved, with severe abdominal pain.²⁵ If surgery is done in these cases for suspected appendicitis, the biopsied nodes show the microscopic pathology of cat scratch fever. Mediastinal lymphadenopathy, with symptoms of primary atypical pneumonia, has been reported in cases of cat scratch disease.²⁶ An anginal form of the disease also occurs, with involvement of the pharyngeal and tonsillar lymphatic tissues.^{25,27} Osteolytic lesions have been reported twice, once in the ilium²⁸ and once in the femur.²⁹

LABORATORY PROCEDURES

As there are no diagnostic tests for cat scratch fever, laboratory studies are used to exclude other diseases in the differential diagnosis. The findings are not remarkable, except for slight elevations of the sedimentation rate and white blood count. Instances of leukocytosis or leukopenia are rare. The lymph nodes and aspirated pus are sterile on culture for fungi, bacteria, and viruses. Neither intracellular nor extracellular microorganisms can be demonstrated in direct

smears of the pus or in sections of the biopsied nodes. Further negative tests include the Frei, tuberculin, heterophile antibody, and blood Wassermann tests and agglutination tests for tularemia and brucellosis. Of limited value is the complement fixation test against *Lygranum*, which is positive in low titers late in the course of the illness in about one-half of the cases.

SKIN TEST

Antigen prepared from the pus aspirated from the nodes of patients with cat scratch disease produces a typical tuberculin type reaction in other cases of cat scratch disease and in about 5% of healthy persons.³⁰ There is no commercial antigen. There is an acute shortage of cat scratch fever antigen throughout the country because it is being more widely used in the differentiation of undiagnosed lymphadenopathies. When a case is suspected, the lymph nodes should be aspirated to aid in the diagnosis and some of the pus should be frozen so that antigen can be prepared from it.

Pus is aspirated from a suppurative node and tested by the use of cultural methods to demonstrate that it is sterile. It is then diluted 1 to 5 with isotonic, sterile, pyrogen-free saline and heated at 60° C. for one hour on two consecutive days. The antigen is then retested for sterility. Its specificity is checked by testing on known reactors and on negative controls. The antigen is stored frozen at 0° C.

The skin test is performed by injecting intracutaneously 0.1 cc. of the well shaken antigen. An immediate, nonspecific, histamine type of reaction, which fades in six to twelve hours, occurs in most patients. At twenty-four to forty-eight hours, a tuberculin type of delayed hypersensitivity develops. A positive reaction is one in which an area of induration or erythema develops that is 6 mm. or greater in diameter. On occasion, the skin test may undergo ulceration and require weeks or months instead of days for its involution.³¹ Occasionally, at the site of a strongly positive test, an area of brown pigmentation forms that may last a month or longer. Skin test reactivity may persist for as long as twenty-eight years.³²

Although about 5% of control patients tested give positive skin tests to the specific antigens, certain groups, such as veterinarians, members of families of patients with cat scratch disease,³⁰ and patients with nonspecific urethritis,³³ show an incidence of positive skin tests 3 to 6 times greater than the general population. We believe that these positive skin tests result from subclinical infections in the first groups and that, in

the patients with nonspecific urethritis, they represent either cross reactivity with a related virus or cases of cat scratch disease with urethritis as another of the protean manifestations of the illness.

PATHOLOGY OF LYMPH NODES

Hyperplastic lymph nodes removed early in the course of the disease are usually discrete and freely movable, beef-red in color, and firm in texture and show punctate, pale areas on cut section. Microscopically, there is reticuloendothelial proliferation and hypertrophy of the germinal centers. Reticular cells aggregate in various germinal centers and develop into epithelial cell granulomas. Lymphocytes, plasma cells, eosinophils, and neutrophils are present about these epithelial cells. At this stage, the pathology resembles that found in sarcoidosis and giant follicular lymphoma.

Nodes removed later in the disease show loss of color and firmness. Cut sections show punctate, pale areas and many discrete, pale yellow areas, oval in shape, varying in size up to several millimeters in diameter and exuding purulent material. These microabscesses are seen on microscopic examination to have formed by enlargement of the epithelial granulomas, their infiltration with neutrophils, and their subsequent central necrosis. The dead neutrophils and remnants of the epithelial cells are gradually converted to a homogeneous eosinophilic material. A well defined zone of epithelial cells frequently surrounds the microabscesses and is in turn surrounded by a cellular reaction characterized by lymphocytes and plasma cells. This picture is similar to that of suppurative lymphadenitis, tularemia, brucellosis, and lymphogranuloma venereum.

The final stages of the disease find the lymph nodes matted together and adherent to the surrounding tissues. They are pale, mottled, and gray in color, soft and friable in texture, and at times converted almost entirely to pus. These nodules tear so easily that their identifiable remnants may be only brownish-red hyperplastic nodules, adherent to each other and the nearby inflamed tissues. Microscopically, the microabscesses are observed to have enlarged by direct extension and by fusion with adjoining abscesses. Nuclear degeneration and loss of cytoplasm occur in the epithelial cells, and cells of the Langhans giant type form. Fibroblastic proliferation and collagen formation are apparent in the surrounding tissues. Such a caseous node resembles that found in bovine tuberculosis.^{13,18,19,34}

These 3 stages of the pathologic process usually coexist in the excised nodes, together with all types of transition between them.

DIAGNOSIS

Until the causative agent is known and there are methods for its laboratory identification, the following criteria are necessary for the diagnosis of cat scratch disease. For typical cases, criteria 1 and 3 of the next 4 are necessary; for atypical cases, all 5 are essential.

1. Regional lymphadenopathy.
2. History of cat contact, especially of cat scratch or cat bite.
3. Biopsy of the enlarged nodes and finding of microscopic picture consistent with that of cat scratch disease.
4. The elimination of all other possibilities. This includes obtaining a sterile culture from the biopsied lymph node or pus aspirated from it and ruling out by specific tests tuberculosis, tularemia, brucellosis, infectious mononucleosis, lymphogranuloma venereum, and, where necessary, malignant diseases such as Hodgkin's disease, lymphoma, and lymphosarcoma.
5. A positive skin test to cat scratch disease antigen. There are at least 2 strains of the infectious agent causing cat scratch disease, and there may be more.³⁵ Rare cases of cat scratch disease will give a negative skin test to proved antigen. They will, however, react to other cat scratch disease antigens with which they are

tested.⁸ Therefore, in cases in which the other diagnostic criteria are met and there is a negative skin test, a second test antigen should be used. If a negative test is obtained to the second antigen, the case should be considered *not* to be cat scratch fever. A positive skin test has a confidence level of 95% except when another member of the patient's family has had cat scratch disease in the past, or the patient is a veterinarian or an animal hospital employee. When positive in such patients, it has a confidence level of only 75%.

THERAPY

There is no antibiotic, chemotherapeutic, or medical treatment that alters the course of cat scratch fever. Since the illness is usually benign and self-limited, no treatment is usually required. Where treatment is necessary because of the severity of the symptoms or because of prolongation of the illness, it should consist of surgical removal of the enlarged nodes^{34,36} or, in cases of Parinaud's oculoglandular syndrome, of excision of the retrotarsal granuloma.²⁰ Such therapy causes prompt remission of the local and systemic reaction. In milder cases, single or multiple aspirations of suppurative nodes will often shorten the illness and promote regression of the adenopathy. When this is done, the material aspirated may be used for diagnostic studies and should also be preserved so that skin test antigen may be prepared from it.

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RESERPINE-TREATED CATS have more and greater tissue injuries, including perforations, than do untreated controls after esophageal perfusion with human gastric juice.

Possible causes of the decreased tissue resistance are (1) increased ACTH release through hypothalamic stimulation with resultant corticosteroid secretion, (2) serotonin release, and (3) local factors such as vascular spasm or decreased blood flow because of hypotension.

Among 19 reserpine-treated cats with either esophageal segments or the entire esophagus perfused with human gastric juice, 4 perforations occurred, compared to none in 19 controls. A significant increase in grade of digestion was noted in specimens from 15 of the animals given reserpine.

A. S. LEONARD, W. O. GUILLEN, and O. H. WANGENSTEEN: Experimental preparation showing effect of reserpine on tissue resistance. *Proc. Soc. Exper. Biol. & Med.* 103:190-192, 1960.

Idiopathic Tropical Eosinophilia

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IN 1923, De Langen and Djamil¹ described 85 per cent eosinophilia with asthmatic symptoms. Subsequently, Armand-Dellille and De Pierdon² in 1927, Bass³ in 1931, Burke and Gupta⁴ in 1934, and Benjamin and Barton⁵ in 1937 published their papers on the subject. In 1940, Frimodt-Møller and Barton⁶ described the cases of 175 patients who complained of cough, hemoptysis, loss of appetite associated with characteristic x-ray findings and high eosinophilia and grouped them under the term "pseudo tuberculosis associated with eosinophilia." In 1941, Alexander⁷ termed a small number of similar cases "intrinsic asthma." Weingarten,⁸ in 1943, called the condition a "new disease entity" and suggested the name "tropical eosinophilia" after reviewing the literature. In 1950, Ball and Tren⁹ reviewed the literature and reported 100 cases and suggested that the condition should be called "tropical pulmonary eosinophilia."

Aitken and Roy,¹⁰ in 1953, reported cases in which organs other than lungs were attacked and made a plea to delete the word "pulmonary" and were in favor of the term "eosinophilic syndrome group." Gelfand,¹¹ in 1956, reported 2 cases without pulmonary involvement.

A wide review of the literature shows that considerable confusion exists about the nomenclature of the condition. As it is fundamentally a condition of eosinophilia, a list showing causes of eosinophilia is given below:

Conditions associated with eosinophilia

1. Tropical pulmonary eosinophilia
2. Cases reported in literature under various names
3. Allergic states, no allergen

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4. Löffler's syndrome
5. Allergic states of known allergens
6. Familial eosinophilia
7. Eosinophilic leukemia
8. Periarthritis nodosa
9. Eosinophilic granuloma
10. Helminth infection
11. Hodgkin's disease
12. Liver diet in pernicious anemia
13. Chronic myeloid leukemia
14. Pulmonary coccidioidomycosis
15. Skin disease
16. Convalescence from acute infection
17. Following irradiation
18. Following streptomycin treatment in tuberculosis
19. Scarlet fever, acute stage
20. Neoplasms
21. Infectious mononucleosis

It is evident from the foregoing list that conditions No. 9 through 21 are clinical entities. In 1952, Crofton and associates¹² suggested including No. 8 with No. 1 to 5 of the list. We feel that periarthritis nodosa is a distinct clinical entity and belongs under collagen diseases.

Eosinophilic leukemia (No. 7) is associated with splenomegaly. Though some workers have recorded splenomegaly in some of their cases of tropical eosinophilia, most investigators have not. We did not come across any enlargement of the spleen in our series.

Recorded cases of familial eosinophilia (No. 6) are asymptomatic. From a review of the literature and our own observations, it seems that heredity has no influence on the condition.

The allergic states caused by known allergens (No. 5) are excluded from this group, as the etiology of the condition under discussion is unknown.

It is by no means clear from clinicopathologic evidence how exactly tropical eosinophilia differs from Löffler's syndrome (No. 4). It may be a different manifestation of the same pathologic process. We have no record of a case of typical Löffler's syndrome in our series. As the etiology and pathology of this syndrome are not definitely established, we think that the two conditions should be studied together.

We have seen cases of massive eosinophilia

with allergic manifestations due to unknown allergens, with or without pulmonary findings, which respond to the treatment in the line of tropical eosinophilia. These cases in our opinion should be included in the group of tropical eosinophilia.

As cases have been recorded without any pulmonary symptoms and signs, the word "pulmonary" should be deleted from the nomenclature. In our opinion, the nomenclature for these conditions should be "idiopathic tropical eosinophilia."

Idiopathic is an addition to the most widely used nomenclature at present. Eosinophilia due to various tropical diseases, notably helminthiasis is quite common. The term "tropical eosinophilia" is not clear enough to exclude these conditions of eosinophilia with known etiology and tends to create confusion. By prefixing "idiopathic," this is avoided. Besides, the word idiopathic does not preclude further investigations to find the causative agent or agents. Our idea of adding "idiopathic" is a stopgap as well as a stimulus. It will be dropped as soon as the etiologic agent or agents are found, and, in the meantime, it will remind one that the disease is still idiopathic and remains to be explored.

MATERIALS AND METHODS

We are presenting here the clinical features of 120 cases of idiopathic tropical eosinophilia, including 40 cases studied by one of us (N.L.) in Calcutta in 1950. The remaining 80 cases include those studied in Dacca Medical College Hospital and Mitford Hospital and personal cases.

Study of clinical pathology, bone marrow, and lymph node biopsy of a limited number of cases are presented. Lung biopsy of one untreated case has been studied.

In presenting our findings, we have as far as practicable reviewed the literature and covered the subject from clinical features to treatment.

CLINICAL FEATURES

Age incidence. The minimum age recorded in the series is 1 year, the maximum 65 years, with the maximum incidence occurring during the third and fourth decades (figure 1). This conforms with most of the published literature.¹³⁻¹⁶

Sex. Of 120 patients in the series, 110 were males and 10 cases were females. Thus, the disease was 11 times more common in males than in females in our series.

Occupation. There seems to be no relation between the incidence of the disease and occupation. Our patients belong to all socioeconomic

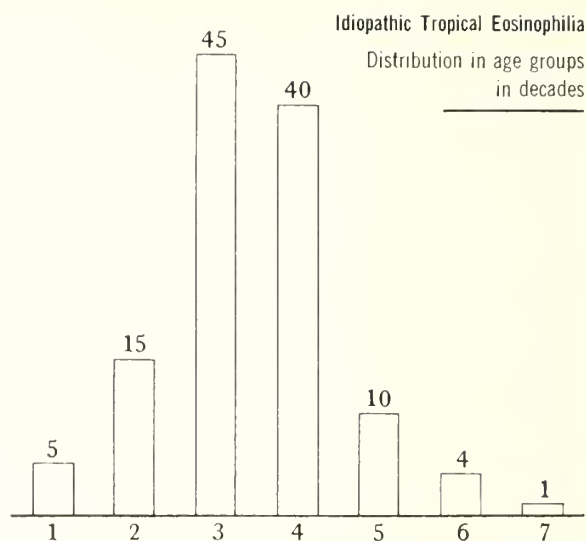


Fig. 1.

groups from farmers to administrative officers without significant difference.

Heredity. Bronchial asthma has a definite hereditary trend recorded in the first book on the subject by Sir John Floyer¹⁷ in 1698. We have no evidence of a hereditary factor in our series. A second member of a family with clinical features of tropical eosinophilia has never attended the clinic. Weingarten⁸ did not find any familial or occupational incidence in a study of 81 cases.

Seasonal incidence. In 1950, Ball and Treu⁹ reported greater incidence of tropical eosinophilia during rainy seasons and showed the parallelism between the rainfall and the incidence of the disease. In 1953, Misra and associates¹⁸ reported that the incidence was high from October to March. Our cases were reported throughout the year, and we did not find any seasonal prevalence.

Race. All our cases were Indo-Pakistanis—40 Indians and 80 Pakistanis.

SYMPTOMATOLOGY

Livingstone¹¹ and Viswanathan¹³ divided the condition into two different types, acute and chronic, and pointed out that, if left untreated, some proportion of the cases clear up completely while others advance to the chronic stage.

In this series, the time interval between the appearance of symptoms and the attendance at the clinic varied from ten days to eighteen years. Patients reported increases in the development of symptoms in the first few years, with a gradual decline.

This might be explained by the lack of consciousness and laboratory facilities on the part

TABLE 1
PULMONARY EOSINOPHILIOSIS,
SYMPTOMATOLOGY IN 207 CASES

Symptoms	No. of cases	Percentage
1. Cough	201	98.1
2. Lassitude	182	88.6
3. Breathlessness	176	85.02
4. Loss of weight	113	84.5
5. Low, intermittent fever	91	43.9
6. High, remittent fever	6	2.6
7. High, continuous fever	5	2.4
8. Asthma	56	27.05
9. Heaviness, chest pain	26	12.5
10. Hemoptysis	9	4.3
11. Palpitation	6	2.08
12. Enlarged spleen	74	35.7
13. Enlarged glands	2	0.9
14. Positive x-ray findings	105	50.7

TABLE 2
IDIOPATHIC TROPICAL EOSINOPHILIA,
SYMPTOMATOLOGY IN 120 CASES

Symptoms	No. of cases	Percentage
1. Cough		
Nonproductive	35	27.5
Scanty expectoration	35	27.5
Moderate expectoration	10	8.3
Paroxysmal		
Simulating bronchial asthma	26	21.6
Simulating status asthmaticus	4	3.3
2. Dyspnea	38	31.6
3. Hemoptysis		
Streaks	26	21.6
Frank	6	5
4. Sneezing, excessive	2	1.6
5. Fever		
99 to 101° F.	37	30.8
102 to 104° F.	4	3.3
6. Loss of weight	18	15
7. Chest pain, vague	39	32.5
Pleuritic	1	0.8
8. Anorexia	11	9.1
9. Angioneurotic edema	3	2.5
10. Physical signs		
Hyperresonance on percussion	17	14.1
Harsh breath sounds with prolonged respiration	32	26.6
Rales	11	9.1
Rales and bronchi	20	16.6
Enlarged spleen	0	0
Enlarged lymph nodes	1	0.8

of general practitioners, who, on the basis of symptomatology, diagnosed such conditions as common cold, muscular pain, allergy, tuberculosis, psychosis, and, finally, bronchial asthma.

Only 4 of the 120 patients came to the clinic with a temperature of 102 to 104° F. This was not the onset of the disease. Because of the factors previously described, very few cases are likely to be detected in the early stage unless blood examinations are done as a routine procedure. In our experience, most of the cases were subacute in onset and passed on to the chronic stage. Acute exacerbation, however, oc-

curs, as we have seen 4 cases simulating status asthmaticus and also the same number of cases with high fevers of 102 to 104° F.

We are, therefore, not in a position to discard totally the clinical classification of Viswanathan, as formerly described, but emphasize that subacute onset is an entity and is more common than the acute onset. At the same time, acute exacerbations of chronic cases do occur, such as exaggerated dyspnea, status asthmaticus, high fever, and so forth. This conforms with the finding of Frimodt-Møller.¹⁵ The occurrence of signs and symptoms has been tabulated in tables 1¹³ and 2.

Cough. This is the most common of all symptoms. Of 120 patients, 35, or 27.5 per cent, had nonproductive cough. In 35, or 27.5 per cent, cough resulted in scanty expectoration and in moderate expectoration in 10, or 8.3 per cent. Cough was paroxysmal and nocturnal in nature in 26 patients, or 21.6 per cent.

Dyspnea. Next in frequency is dyspnea of variable degree from exertional to status asthmaticus. In our series, 4 patients, or 3.3 per cent, were admitted to the hospital in a state of status asthmaticus.

Chest pain. Vague chest pain has been described as having an incidence from 12.5 per cent¹³ to 50 per cent.¹⁶ The incidence in our series is 32.5 per cent. Only 1 patient had pain simulating pleural pain. Viswanathan¹³ and Khan,¹⁶ however, did not describe the pain in the chest. The pleuritic pain may be explained by pleural adhesions described under thoracotomy findings.

Hemoptysis. In our series, 26 patients, or 21.6 per cent, revealed a history of streaks of hemoptysis, and only 6, or 5 per cent, gave a history of frank hemoptysis.

Other symptoms. The other complaints were sneezing in 2 patients, or 2.6 per cent; slight fever in 37, or 30.8 per cent; loss of weight in 18, or 15 per cent; anorexia in 11, or 9.1 per cent; and angioneurotic edema in 3, or 2.5 per cent. Mention must be made of the cases with the symptoms of sneezing and angioneurotic edema. The 2 patients with the former reported only with colds accompanied by sneezing, and 2 of the 3 with angioneurotic edema had only this symptom.

As far as physical signs are concerned, the most common finding in the series was harsh breath sounds with prolonged respiration, associated in most cases with rales of variable extent over the chest. Evidence of emphysema, namely, hyperresonance on percussion, was found in 17, or 14.1 per cent of cases.

Splenomegaly and lymphadenopathy have been reported by various authors. Patel¹⁹ found lymphadenopathy in 45 per cent of his cases, and Viswanathan¹³ found this condition in 0.1 per cent of his cases. Jhatakia²⁰ reported splenomegaly in 3 of 146 cases, and Viswanathan²¹ found the evidence to be as high as 30 per cent. However, D'Abrera and Stork²² and Misra and associates¹⁸ did not find hepatomegaly, splenomegaly, or adenopathy among their cases. Our findings corroborate with the last authors. We recorded no case of hepatomegaly or splenomegaly. Only 1 patient in our series had enlarged lymph nodes.

TABLE 3
RADIOLOGIC FINDINGS VERSUS EOSINOPHIL COUNTS,
PER CU. MM. IN 36 CASES

Streaky lung fields	Patchy opacity	Heavy hilar shadow	Mottling both sides	Specific infiltration	Nil
5,616	4,080	2,700	5,040	19,240	1,200
7,840	7,128	5,464.8	5,200		1,260
8,100		6,240	5,670		1,680
8,320			6,600		3,200
13,261.5			7,260		4,060
13,440			8,400		4,600
17,400			8,400		7,600
27,930			10,450		21,280
			12,600		26,000
			13,510		
			14,670		
			21,760		
			31,200		
Total 8 (22.2%)	2 (5.5%)	3 (8.3%)	13 (37.2%)	1 (2.7%)	9 (25%)

We have been able to collect the radiologic reports and the simultaneous eosinophil count in 36 cases (table 3). Only cases in which we agreed with the radiologic report and had the simultaneous blood count are included. In 8 patients, or 22.2 per cent, streaky lung fields or increased bronchovascular markings were noted, with corresponding eosinophil count per cu. mm. ranging between 5,616 and 27,930. Patchy opacity was present in 2 patients, or 5.5 per cent, with an eosinophilic count between 4,080 and 7,128 per cu. mm. Heavy hilar shadows were seen in 3 patients, or 8.3 per cent, with an eosinophilic count of 2,700 to 6,240 per cu. mm. Mottling on both sides appeared in 13 patients, or 37.2 per cent, with an eosinophilic count of 5,040 to 31,200 per cu. mm.

In 1 case, radiologically specific infiltration was doubted, and, in 9 cases, or 25 per cent, there was no radiologic abnormality. This means that about a quarter of the cases with variable eosinophilic counts showed no radiologic abnormality. Comparison between the eosinophil

count and the radiologic finding shows no clear-cut relationship, for example, 26,000 eosinophils per cu. mm. of blood showed no radiologic change, whereas 5,040, 5,616, 7,128, and 19,240 eosinophils per cu. mm. produced mottling on both sides, streaky lung fields, patchy opacity, and shadows suggesting specific infiltration, respectively. Viswanathan²¹ reported positive x-ray findings in 50.7 per cent of his cases.

Lung biopsy. There is a postmortem record of a patient with tropical eosinophilia who died of arsenical encephalopathy.²¹ We could not find any record of lung biopsy in this condition. As a postmortem examination of a patient with tropical eosinophilia is a mere chance and also because the superadded cause of death may alter the actual pathology of tropical eosinophilia, we decided to carry out lung biopsies of such cases. We have done a lung biopsy of an untreated case of a patient whose total eosinophilic count was 26,000 per cu. mm.

During thoracotomy, it was observed that widely scattered nodules resembling miliary tubercles were present over the lung surface. The size of these tubercles varied from 1 to 3 mm. in diameter. There were about 6 very thin avascular fibrous adhesions between the visceral and parietal pleurae. A biopsy of approximately 1½ in. by ½ in. was taken from the base of the right lung. Histologically, it showed nodular areas of consolidation in which the alveoli were filled mostly with cellular exudates. The predominant cells were eosinophils. There was no inflammatory exudate in the bronchioles. In between the nodules, the lung tissue showed emphysematous changes. These alveoli were empty. There was marked infiltration of eosinophils and a fibroblastic reaction of a moderate degree in the inter-alveolar septa. Areas of hyalinization were scattered all over. No giant cell was present, such as was found by Viswanathan²¹ in his postmortem of a case of tropical eosinophilia and also by Bayley and associates²³ in their postmortem of a case of Löfller's syndrome. There was no pathologic change in the wall of the blood vessels. The lumens were filled with red blood cells and plenty of eosinophils. The pleura showed no pathologic change.

Lymph-node biopsies. Viswanathan²⁴ and Khan¹⁶ have reported that enlargement of lymph nodes is an occasional finding in this condition. We have also recorded a case. We performed lymph node biopsies in 4 cases. The histologic picture showed neither marked hyperplasia nor any change in the architecture. There was an infiltration of eosinophils in the pulp of varying number. There was no evidence of any localized

accumulation of eosinophils nor was there any hyalinization, fibrosis, or giant cell.

Bone marrow. Viswanathan²⁴ reported an increase in mature eosinophil cells in the marrow without any significant rise in the number of precursors of eosinophils. We studied sternal puncture material of 10 cases and found a marked increase in eosinophilic myelocytes together with mature forms.

LABORATORY INVESTIGATION

Cell count. The highest leukocytosis we have recorded in our series is 40,000 per cu. mm.

The eosinophil counts in all our cases were over 2,000 per cu. mm. except 3 cases in which the eosinophil counts were 1,200, 1,260, and 1,680 per cu. mm., respectively. These we included in the list because of their symptomatology, absence of a known etiologic factor, and clinical response to treatment for idiopathic tropical eosinophilia.

Hemoglobin. Most of our cases showed a slight to moderate degree of low hemoglobin content. However, the cause of anemia was not investigated.

Erythrocyte sedimentation rate. In most of our cases, the erythrocyte sedimentation rate obtained by the Westergren method was high, ranging between 3 to 65 mm. in the first hour. Only 10 cases showed an erythrocyte sedimentation rate below 10 mm. in the first hour. The high rate, however, came down with improvement of the clinical condition.

Sputum. After examination of 30 sputums for eosinophils, we are of the opinion that there is no correlation between the number of eosinophils in the sputum and blood. Mites were not found in any of the specimens we examined.

Weil-Felix test. Menon²⁵ likes to use the Weil-Felix test for diagnosis and prognosis of this condition. Of 14 cases, we found 1 positive, which again revealed a definite history of exposure.

Cold agglutination test. The result of cold agglutination tests published by Viswanathan and Natarajan²⁶ in 1945 and Ball and Treu⁹ in 1950 differ widely. Cold agglutinin was absent in 8 of our cases. This conforms with the findings of Ball and Treu.⁹

Paul-Bunnell test. Using this test, Viswanathan¹³ recorded 3 positive cases out of 9. The results of absorption tests are not recorded. We have done Paul-Bunnell tests in 6 cases, 4 of which were positive in a titer of 1/160 to 1/320. Our absorption tests of 2 cases showed that the antibody is completely absorbed by guinea pig kidney suspension and partially absorbed by ox-cell suspension.

Complement fixation test with filarial antigen. Danaraj²⁷ has reported that in all of 65 cases of tropical eosinophilia, complement fixation tests with filarial antigen were positive in high titer and declined after treatment. Negative results have been recorded in mild eosinophilia with pulmonary symptoms. We have found positive complement fixation tests with filarial antigen in all of our 16 cases so far examined. The results are very encouraging. The tests may prove very useful in the early diagnosis and prognosis of some of these cases.

It may be mentioned here that 13 of 22 cases of *Strongyloides stercoralis* infection showed positive complement fixation tests with filarial antigen.

Complement fixation test for kala-azar with Kedrowsky's antigen. Banerjee²⁸ stated that this test was positive in 5 per cent of his cases of tropical eosinophilia. We tested the sera of 15 of our patients, and results were negative in all cases.

ETIOLOGY

Danaraj²⁷ postulated filarial etiology of idiopathic tropical eosinophilia from the positive complement fixation test with high dilutions and its decline after diethylearbamazine therapy. We found similar results in all of our 16 cases. An experiment by Buckley²⁹ in 1958 in a human volunteer with inoculation of *Wuchereria malayi* larvae of monkey origin and *W. philangii* of cat origin is of great interest and should stimulate further interest in that direction.

We failed to demonstrate any structure suggestive of any portion of the nematode in our lung and lymph node biopsies.

TREATMENT

Arsenic was introduced for the treatment of eosinophilia by chance in 1943 by Weingarten,⁸ who, while treating a patient suffering from syphilis with arsenic, found that there was rapid improvement of symptoms and reduction of eosinophilia. Since then it has been widely used with success. One of us³⁰ studied the effect of arsenic in 1952 in 40 patients with uniformly good results. In 1950, Ball and Treu⁹ reported the effect of arsenic in 92 patients with similar results. They, however, mentioned occasional exacerbation of symptoms, which has subsequently been confirmed. We conducted treatment in 54 patients of 80 cases studied in East Pakistan—20 with arsenicals, 20 with Hetrazan, 10 with prednisolone, and 4 with antihistamines.

No serious toxic reaction was observed in any group. Antihistamines were not continued in

more than 4 patients, as the response was not satisfactory and no satisfactory response to antihistamines for this disease has been recorded.

Organic arsenicals have been enjoying the widest field until very recently. Diethylcarbamazine was introduced by Friess and associates³¹ in 1953 when they found *Wuchereria malayi* in the lymph nodes of 6 patients.

Reports by Wildervanck and associates³² in 1953, Ganatra and Lewis³³ in 1955, and Chaudhuri³⁴ in 1956 also have been favorable about diethylcarbamazine therapy. Danaraj³⁵ recently treated 110 patients without any past or present history of filariasis. He suggested treatment with 6 mg. of diethylcarbamazine per kilogram of body weight thrice daily for five days. Of 110 patients, 3 relapsed in six months. There were no appreciable toxic reactions.

In our series of 20 patients followed from two months to one year, only 2 relapsed, and we can report that they responded with reinstitution of therapy.

Of the arsenicals, we used only 0.78 gm. of diethylaminoacetarsol (Acetylarsan) biweekly for four to six weeks. There were 4 relapses, and 1 patient showed no response. The 4 relapses were in patients in whom we could continue treatment for four weeks only. We, however, recorded no untoward symptoms.

Danaraj³⁵ considered toxic reactions to arsenicals more common than published reports lead one to believe and laid stress on the occurrence of encephalopathy, a severe reaction for such a mild disease and one that has proved fatal. The same author has recorded fatal encephalopathy after treatment with neoarsphenamine. Since diethylaminoacetarsol proved the least toxic in our experience with 40 cases treated in 1950, we advocate treatment with this arsenical.

Prednisolone was used in 10 patients in our series. The response in these cases was most rapid. The symptoms improved, and total white cell count and the eosinophil count showed a significant fall within twenty-four to forty-eight hours. All patients initially responded equally to value. Relapse occurred in 2 patients in whom treatment was stopped suddenly. This can be taken to mean that, in treating eosinophilia with this drug, the same principle must be followed as in other conditions, that is, gradual withdrawal of it.

DISCUSSION

Although idiopathic tropical eosinophilia of late has evoked much enthusiasm among workers in the field with encouraging results, much about the disease has yet to be explored. Clinical as-

pects have been most extensively studied, with special reference to respiratory involvements, possibly because physicians are aware of the prevalence of tuberculosis in the tropics and the clinical and radiologic similarity of the two conditions in a good proportion of cases. Tragedies of multinomenclature from "pseudo tuberculosis" to "eosinophilic syndrome group" clearly depict the amount of confusion the condition has created. Aitken and Roy¹⁰ and Gelfand¹¹ have recorded cases in which organs other than lungs were involved. We have had 4 similar cases in patients who came to the clinic solely with extrapulmonary symptoms, namely, sneezing and angioneurotic edema. Accordingly, we agree with these authors that the word "pulmonary" should be deleted and recommend the word "idiopathic" as a prefix to differentiate the condition from other known tropical entities associated with eosinophilia.

We have recorded cases in patients 1 to 65 years of age, with a maximum incidence during the third and fourth decades. We have found the disease 11 times more common in males than in females. It is not related to occupation or season. Records of the occurrence of idiopathic tropical eosinophilia in more than 1 member of the same family, which some workers have reported, does not establish hereditary influence of disease. We have recorded no second case in the family. Danaraj³⁵ reporting a greater prevalence of the disease among the Indians in Malaya than among the Malaysians and the Chinese in Malaya, wanted to show racial influence. Our study was limited to West Bengal in India and Dacca in East Pakistan, and all our patients were Indio-Pakistanis. Unless a detailed study is done on this issue, we are not in a position to comment.

The disease has three modes of onset, namely, acute, subacute, and chronic, the first two passing into the chronic stage and the subacute being the most common. This is in contradistinction to two clinical types previously described—acute and chronic. Cough, dyspnea, chest pain, and hemoptysis are the most common symptoms. Enlargement of lymph nodes is a rare finding. We have recorded only one such case. We think that splenomegaly is not a feature of the disease. Enlargement of the spleen recorded previously is, in our opinion, a coincidental finding unrelated to this disease. This, however, requires further elucidation by study of the spleen, for example, by biopsy or puncture.

Radiology as a diagnostic aid has limitations. A quarter of our cases showed no radiologic abnormality. Again, the four categories into which

we have divided the radiologic findings—mottling on both sides, streaky lung fields, heavy hilar shadows, and patchy opacity—are found in many other diseases. The only way of diagnosing idiopathic tropical eosinophilia is by suspicion and routine blood examination in cases in which symptomatology and roentgenograms of the chest suggest the possibility of the disease. Other conditions producing eosinophilia do not cause much difficulty. Helminthic infections, which are the most common of these conditions, can be detected by repeated stool examinations. Moreover, we have not come across a high eosinophil count as a result of helminthiasis. Bronchial asthma does not produce any radiologic change as previously described and neither leukocytosis nor high eosinophilia present. Also, bronchial asthma is definitely influenced by heredity in about 40 per cent of cases. Löffler's syndrome has been described by Engel³⁶ and Harkavy³⁷ as being due to a variety of allergens, though according to Löffler³⁸ himself, it is due to the pulmonary phase of ascariasis and characterized by fleeting shadows in the lung. Until a significant number of cases of this group is studied in detail, including lung biopsy, we are not happy to recognize it as an entity distinct from idiopathic tropical eosinophilia.

Lymph nodes are undoubtedly infiltrated by eosinophils at some stage of the disease, at least in some cases, but the incidence of enlargement is very low. The fundamental changes in the lungs are areas of miliary consolidations, interstitial pneumonitis, and compensatory emphysematous areas without any visible change in the bronchioles. It would appear to the authors that hyalinization is also of importance in this condition.

Anemia, high erythrocyte sedimentation rate, cold agglutinin, Paul-Bunnell test, and the relevant biochemical changes require much to be explored. We would suggest a detailed biochemical study and biopsy of each and every accessible organ.

Unfortunately, there is as yet no general agreement on the lowest limit of an eosinophilic count which may be taken as a diagnostic criterion of the condition. Many investigators believe that it is 20 per cent. In 1957, it was set as low as 6 per cent in the *British Medical Journal*.³⁹ We strongly advocate expressing the number of different leukocytes as the absolute number per cubic millimeter instead of as a percentage only. Adams and Maegraith⁴⁰ are of the opinion that the total eosinophil count should exceed 2,000 per cu. mm. in this condition. Though most of the cases recorded show an eosinophilic count of

2,000 or more per cu. mm., we have at least 3 cases in our series in which the total eosinophilic count was as low as 1,200 per cu. mm., and we included them because of their symptomatology, radiology, and clinical response.

Eosinophilia of 10 to 15 per cent is quite common in helminthic infection in the tropics and disappears after treatment with anthelmintics alone. Therefore, 6 per cent eosinophilia is too low to be included in the list. We are at present studying the eosinophil response to helminthic infection. Positive complement fixation tests with filarial antigen in all of the 16 patients whom we examined are very encouraging. If further study shows equally significant results, this test may possibly prove to be the most important diagnostic criterion. Very encouraging results with diethylcarbamazine, positive complement fixation tests with filarial antigen, and a recent study by Buckley²⁹ point toward filarial etiology. If this is so, what is the species? Could there be more than one etiologic agent? Are they antigenically the same with filaria?

SUMMARY

1. Literature on tropical eosinophilia has been reviewed.

2. A new nomenclature, idiopathic tropical eosinophilia, has been suggested and reasons why it should be adopted are given.

3. Clinical features of 120 cases in patients ranging from 1 to 65 years of age have been described and compared with the findings of other authors.

4. Relationship between eosinophil counts and radiologic findings has been described.

5. Clinical pathology and the study of bone marrow in a small number of cases as well as the histopathology of a case of lung biopsy have been described.

6. Probable etiology has been discussed.

7. A preliminary report on the treatment of idiopathic tropical eosinophilia with arsenicals in 60 cases, diethylcarbamazine in 20 cases, prednisolone in 10 cases, and antihistamines in 4 cases have been reported.

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LEUKEMIA MAY BE provoked by radiation therapy. However, a considerable amount of irradiation usually is necessary to produce the disease in adults.

Generally, a latent period elapses between the time of irradiation and appearance of leukemia. This is sometimes only one year but more often is three to ten years.

Onset may simulate aplastic anemia. In such cases, diagnosis is verified by bone marrow examination or biopsy.

To help establish criteria for diagnosis, all cases of leukemia resulting from irradiation should be published. Information regarding dosage and character of the radiation exposure is especially necessary.

W. C. MOLONEY: Leukemia and exposure to x-ray: a report of 6 cases. *Blood* 14:1137-1142, 1959.

The 1960 White House Conference on Children and Youth

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PRESIDENT EISENHOWER directed that a White House Conference on Children and Youth be held in March 1960. Such conferences have been held approximately every ten years since 1909, when President Theodore Roosevelt personally invited 217 national leaders to a conference to consider problems of dependent children. That conference was instrumental in speeding child labor legislation and also led to the establishment of the Children's Bureau. In that year, automobiles were beginning to come into general use on the nation's dusty roads. Blériot made the first airplane flight across the English channel, skimming above the waves at 40 miles per hour. In the same year, Peary was the first to reach the North Pole. The phonograph, invented by Edison, was a national fad, with its thready, tinny tunes.

Each succeeding conference has grown in size and scope and has changed in emphasis. The purpose of the 1960 conference, as stated by President Eisenhower, was to promote opportunities for children and youth to realize their full potential for a creative life in freedom and dignity. The more than 7,000 delegates to the 1960 conference were chosen from governor's committees in each state and territory and from a number of national organizations. There were 500 foreign guests from 73 countries.

The National Planning Committee, appointed by the president, worked for two years on physical arrangements, the program, and other details. During that time, each state and territory conducted studies of needs and opportunities of children and youth within its own borders and held so-called little White House conferences to discuss local needs and to make recommendations concerning them. The areas studied in-

cluded education, surveying facilities and needs for the handicapped as well as the gifted child; physical, emotional, spiritual, recreational, and social factors; employment, minority groups, and youth in conflict with the law. Their reports and recommendations were submitted to the national committee for their consideration prior to the White House Conference.

The delegates to the conference represented all categories of citizens. There were 1,000 youth delegates who made an extremely valuable contribution. There were representatives of law, religion, medicine, and other professions. It appeared to this writer that the professions most heavily represented were education, social work, and public health and welfare work. There were 200 physicians at the conference, including the chairman of the program committee, Dr. Philip S. Barba of Philadelphia. The 40 delegates from North Dakota included 6 youth members.

The conference was financed partly by federal funds and partly by foundations and private funds. Congress appropriated \$350,000, and the Ford Foundation, the Rockefeller Foundation, and many others contributed large sums. All delegates paid a \$20 advance registration fee. In North Dakota, and presumably elsewhere, a portion of the delegates' expenses amounting to \$100 was paid for from conference funds. The major portion of the expenses of most delegates was paid by the organization which the delegate represented.

Arrangements for the accommodation and participation of such a large number of people required much planning and coordination. Most delegates felt that this was extremely well done. The opening ceremony was held on Sunday evening, March 27, at the University of Maryland Field House about 15 miles from Washington, the only auditorium in the area large enough to satisfactorily hold such a crowd. Buses took the delegates to and from the University.

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Beginning the next day, Monday, participants were assigned to 5 concurrent daily theme assemblies, to 18 concurrent daily forums, and to 210 concurrent workshops. The assignments were made on the basis of interest in particular problems or aspects of children and youth. The theme assemblies ran for three days from 9:00 A.M. to 10:15 A.M. and consisted of talks by 2 outstanding speakers each day. The daily forums ran from 10:15 A.M. to 12:00 noon, with a panel presentation by 4 or 5 authorities in their field. The workshops ran from 2:30 P.M. to 5:00 P.M., with a designated leader and 30 to 40 participants who discussed particular problems and, by the end of the third day, voted on recommendations. On the fourth afternoon, Thursday, all workshop recommendations were considered at meetings of the complete forum groups, where recommendations were finalized and voted on as the recommendations of the conference. The theme assemblies, forums, and workshops were held in hotels and auditoriums throughout the city, which participants reached by cab or private transportation. On the last morning, Friday, the closing session for all participants was held at the National Guard Armory at the east edge of the city.

Extra activities and functions for delegates included a special concert by the National Symphony Orchestra at Constitution Hall and a tour of the White House. There was a ball with a name band for the youth delegates. Danny Kaye presided at a special evening youth session, which resulted in a number of conference recommendations. Some of the delegates visited the Capitol and called on their representatives in Congress. Many sandwiched in a limited amount of sight-seeing. For physicians, a meeting preliminary to the conference was held on Sunday afternoon at the District of Columbia Medical Society Auditorium. A luncheon meeting for pediatrician delegates preceded that at the Statler-Hilton Hotel. The North Dakota delegation gathered for a group dinner on Sunday evening. A state delegation meeting was held on Thursday morning at the Willard Hotel to exchange impressions of the conference and to make plans for a little White House Conference in Bismarck on May 2.

OPENING SESSION

The opening session at the University of Maryland was highlighted by a thirty-minute speech by President Eisenhower in which he disclaimed any special knowledge of children and youth except that, as a grandfather, he felt that he should be able to speak with some authority. He wished the conference well and expressed

confidence that our young people have the qualities to carry on leadership in their turn. He reminded his audience that hard work and individual responsibility have made this country what it is today. He said that he was concerned for fear our affluent society might tend to make our young people soft and lacking in incentive unless they realize this danger and guard against it. He pointed out some of the challenges facing our nation and the world in the present period of rapid change.

ASSEMBLIES AND FORUMS

The speeches at the theme assemblies and forums beginning on Monday were, for the most part, stimulating and thought provoking. Speaker after speaker emphasized the fact that the weakening of family life is the basic cause of most problems of youth. According to Abraham J. Heschel, Professor at the Jewish Theological Seminary of America, the basic problem of today's youth is today's parent, who has pushed aside his moral responsibilities in his quest for wealth and comfort. We have time for hobbies and watching baseball and for our social activities, he said, but no time to offer companionship and guidance to our children. We delegate parental responsibilities to the school, the church, and social agencies. Material possessions are given precedence over spiritual values. Education is considered as only a means toward power and prestige, not as having true value in itself.

The unsettling effects on youth of increased mobility with consequent uprooting of social and family ties was mentioned by a number of speakers. People move from one part of the country to another, from the city to the suburbs, from the country to the city, or have whole neighborhoods wiped out by new housing developments. According to Lester B. Granger, executive director of the National Urban League, the impact of social change on the attitudes of youth is now evidenced by the lunch counter sit-ins of Negro students in the South as well as by the surge for independence and racial equality throughout the world. He stressed the need for improved community environment, with emphasis on true neighborliness, family workshops, mothers' meetings, recreational facilities, improved school counseling and guidance, and an opportunity for young people to work.

Dr. John H. Fischer, dean of Teachers College, Columbia University, discussed the role of the teacher in helping to shape character as well as to inform the mind. The teacher should encourage his students to search for truth, beauty, honesty, and excellence and to learn that self-

realization and human relationships are more important than outer space. He said that a Negro youth is entitled to the same opportunity for a good education as his white counterpart, and this can only be achieved by racial integration of schools throughout the nation.

A number of us heard speeches and participated in workshop discussions concerning the causes, prevention, and treatment of juvenile delinquency. There was some discussion of the definition of juvenile delinquency, during which one speaker facetiously remarked that a juvenile delinquent is one who has stopped asking you where he came from and now tells you where to go. More seriously, the causes of delinquency are recognized in some cases to be due to hereditary factors and brain damage, but, in the majority of cases, the causes are due to poor home and community environment. Everyone realizes that delinquency does not have its inception when a 15-year-old steals a car and comes into conflict with the law. Such a teen-ager usually has a history of poor elementary school performance, truancy, stealing, or other behavior problems. Some emotional disturbances may even be traced to the preschool age level. Early case finding by the teacher, physician, clergyman, social worker, public health nurse, or parent is essential if delinquent behavior is to be prevented from developing. Treatment of emotional disturbances and behavior problems can usually be handled satisfactorily by community, social, and psychology services and, in more severe cases, by psychiatric evaluation and therapy. A combination of public and private services is the ideal community arrangement. Civic minded citizens need to assess community needs and work together to meet them. Assistance in such planning is available through the National Association for Mental Health, the State Health Department Division of Mental Health, or the United States Public Health Service. School counseling and guidance services, now often lacking or poorly staffed, are an important part of the community program for mental health.

According to Judge Donald E. Long, Circuit Court of Oregon, juvenile delinquency has been increasing steadily for the last ten or twelve years. Delinquency is 4 times more common in boys than in girls, and $3\frac{1}{2}$ times more frequent in urban than in rural youth. He stated that prevention is much more effective than treatment, and the best prevention is a warm home life, recognition, success, and social acceptance as well as consistent discipline and supervision. It was emphasized that as a child grows older, his horizons widen, and community recreational,

social and other resources are important preventive ingredients.

Judge Theodore B. Knudson, Juvenile Court, Minneapolis, stated the need for more trained professional counseling personnel, including probation and parole services, as well as more judges trained and specializing in juvenile court work. Judge Knudson recommended a prehearing conference with any youth involved in juvenile court and usually with his family. The court is then in a position to mete out justice. The youth may be returned home, placed in a foster home, have outpatient psychiatric treatment, or be placed in an institution, with individual treatment best carried out in small units. If considered advisable, the case may be referred from the juvenile court to the criminal court. He described the state-wide juvenile court services now in effect in Minnesota and stated that these have resulted in improvement in handling such cases in that state.

CLOSING SESSION

At the closing session, the speakers were Dr. Ruth A. Stout of Kansas State College and Arthur S. Flemming, secretary of Health, Education, and Welfare. Among other things, both urged federal aid to education. Dr. Stout's premise was that, since the federal government gets 75 per cent of the total taxes of the nation, it should share in the cost of public school education at the local level. Let me hasten to say that this writer does not share that view. Along with millions of other Americans, I believe that personal and community initiative and self-reliance give all of us more to work for and more to live for. Receiving handouts from the federal government, which is already billions of dollars in debt, can only lead to further inflation and constitute another step toward a welfare state. To quote Thomas Jefferson: "If we can prevent the government from wasting the labor of our people, under the pretense of caring for them, they will be happy."

Both speakers stressed the need for spiritual values and ideals, with consequent high moral and ethical standards. Constructive use of the increasing amount of leisure time available for all our citizens was presented as an important challenge. Dr. Stout also spoke of the tensions that are bound to exist between freedom and authority and said that we must confront such tensions and work creatively within them.

CONFERENCE RECOMMENDATIONS

There were 1,600 conference recommendations, 1 for approximately every 4 delegates, many of

which were duplications or contradictions. On a few important issues, however, recommendations were clear. Civil rights and integration of public schools received top priority. A nation-wide study of juvenile delinquency was another recommendation agreed upon. As already mentioned, the recommendation for federal aid to public school education was approved, with the stipulation that control be at the local level.

Local residence requirements for aid to needy families and unwed mothers should be abolished according to another recommendation. States were urged to adopt laws requiring that all illegitimate births be reported to the State Health and Welfare Departments to aid in accurate knowledge of this problem and in making possible adequate aid and counseling for unwed mothers.

Dissemination of birth control information was advised where needed.

Support was given measures to provide adequate housing, education, and working standards for migrant families. Extension of federal minimum wage laws to cover migrant workers was advised. Foreign labor recruitment was decried except on a permanent basis.

An all-out effort was called for to clarify and unify laws concerning marriage, divorce, separation, annulment, and desertion. Strengthened family stability was urged by means of religious influence and counseling programs. An increase of income tax exemptions for child dependents was advised.

Emphasis on sex and crime in our mass communications media was deplored. Television, radio, movies, magazines, and newspapers were asked to accept greater responsibility for transmitting wholesome ideals and values.

These represent only a partial list of conference recommendations and do not include many which will be available in the final conference report. That report will be of interest to those concerned with particular areas or problems of youth.

IMPRESSIONS AND CONCLUSIONS

The question was raised on several occasions as to whether the results of such a conference justified the amount of work, planning, time, and expense involved. An assembly chairman, Mrs. Zelda George expressed it rather clearly when she said that, in such a conference, after all is said and done, more would be said than done. William G. Carr, Executive Secretary of the National Education Association, thought that the scope of the conference was too great to stimulate action on any specific goals for children. On the other hand, the Rev. Phillip A. Potter, Executive Secretary, Youth Department of the World Council of Churches in Geneva, declared that the conference outlook was not big enough. He said that it should focus on needs of children throughout the world, not just in the United States of America. An editorial in the *Washington Post* on the last day of the conference summarized, I think, the feelings of most of the delegates. It was titled "Ten Year Itch" and concluded that the main value of the conference was not in any concrete conclusions reached but in the determination of the delegates to return home and work to help improve opportunities of youth in their own communities and states.

In North Dakota, as elsewhere, this means evaluating and improving, where needed, facilities and services for mental health, physical health and recreation, social and religious programs, juvenile courts, probation and detention, police training, education, school guidance and counseling, youth work programs, care of unwed mothers, adoption, and integration of racial minorities. A Governor's Committee has been organized to follow up and see that the recommendations of the White House Conference are carried out and not forgotten. In this way, and with the cooperation of all interested citizens, President Eisenhower's stated purpose will become a reality, and all children and youth may truly have opportunities for a creative life in freedom and dignity.



Olga Sophie Hansen, M.D.

CATHERINE CORSON WEST, M.D.

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"ISN'T DR. HANSEN funny? He wears a dress and has hair like a lady," remarked 3-year-old Jean upon the arrival of Dr. Olga Hansen as dinner guest of Jean's parents.

"This is my favorite story," says Mrs. Litzenberg.

Dr. Olga Sophie Hansen became Mrs. J. C. Litzenberg on January 27, 1934. Both Dr. Hansen and Dr. Litzenberg, a widower since 1927, had been staff members of the Nicollet Clinic since its beginning in 1921. She had come to the clinic as an associate of Dr. S. Marx White in internal medicine after completing her internship at University Hospital (Elliott Memorial) in 1916. Dr. Litzenberg, an 1899 graduate of the University of Minnesota, had moved his practice from the Donaldson Building to join the Nicollet Clinic as chief of the department of obstetrics and gynecology.

Upon receiving her "Mrs. degree," Dr. Olga became the wife of the president of several national obstetric societies and, thus, took on a new role as hostess. She had a lot of fun with her dual role as "internist-hostess" to obstetric and gynecologic meetings. This pleased Dr. Litzenberg greatly. After his death in 1948, honorary membership in the Central Society of Obstetrics and Gynecology and the Minnesota Society of Obstetrics was bestowed upon Dr. Hansen in appreciation of her husband's and her contributions to these organizations.

Over fourteen supremely happy years together were spent at 717 East River Road, Minneapolis, where Dr. Olga still maintains the Litzenberg residence. Here the various generations of her family congregate from time to time. "All of my children

are O.W.'s, but legitimate," asserts Dr. Olga. In fact, they are one of her many hobbies—3 children, 7 grandchildren, and 2 great-grandchildren.

Stepson Karl Litzenberg is professor of English literature at the University of Michigan. Stepdaughter Avis, Mrs. R. J. Stickney, is unique for her two years' work in Iran as director of women's activities under the Point Four Program of the State Department. Adopted daughter Jeanette, Mrs. E. G. Svendsen, is wife of "Bud" and mother of "Mike" (really Bernard, after Coach Bernie Bierman) Svendsen, each one-time great football centers at Minnesota. Football Saturdays are especially exciting. The backyard is the parking lot, three blocks from the stadium, and, after the game, fellowship and food are provided by Olga Litzenberg.

Dr. Hansen performs all her nonprofessional activities under the title of Mrs. Litzenberg. Her hobbies are travel, Spanish lessons, and, for the most fun in her life, Arthur Murray dancing lessons, with emphasis on the Latin American dances. A regular attendant at the Minneapolis Symphony concerts, she also likes calypso and has toured the Caribbean to hear this music produced by the native islanders.

Balancing this fun, we find that she has always been a serious student. Her record of scholarship in medical school prompted Dr. S. Marx White to answer, "because she was the best man in the class," when he was asked why he chose a woman as his associate in private practice. Dr. White also points out that his mother and his sister had worked hard for women's rights and he was glad to have this opportunity to demonstrate his interest in the equality of women.

Dr. Hansen's association with Dr. White was very timely. In 1915, the year she graduated from medical school, Dr. White returned from Europe. In

CATHERINE CORSON WEST was formerly a clinical instructor in the Department of Radiology and Physical Therapy, University of Minnesota, Minneapolis.

England, he had studied with Sir Thomas Lewis and had brought back with him the second electrocardiograph in Minnesota; the first was at the Mayo Clinic. It was installed in the basement of Millard Hall and grounded to the concrete floor to eliminate extraneous vibrations. "Dr. Hansen went to work on it," says Dr. White, "and became one of the first physicians in Minneapolis to read electrocardiograms." In 1917, she was put in charge of the cardiac clinic at the University Hospital outpatient department and served until 1927. This appointment involved examining patients, reading electrocardiograms, and teaching medical and graduate students in the clinics and the lecture hall.

By the midtwenties, her fame as a student of electrocardiography had extended even into veterinary medicine. Dr. Fitch, University farm veterinarian, called Dr. Hansen to ask "Would it be possible for you to take an electrocardiogram on a decompensated horse?" Dr. Hansen responded and took the 40-lb. portable electrocardiograph to the farm campus. The tracing revealed atrial fibrillation and typical ventricular irregularity. Digitalis was injected in the jugular vein. Another tracing was taken and showed the digitalis effect—rate slowed and ectopic beats. Dr. Fitch inquired of Dr. Hansen, "When would you like to have the autopsy?" The horse was sacrificed, and the postmortem examination revealed endocarditis and old mitral valvular disease. Dr. Hansen reported this "case" to the Minnesota Society of Internal Medicine.

Cardiology, though her chief interest, is almost tied by her interest in diabetes. In 1923, when Banting and Best isolated insulin, patients were still being treated by the Frederick Allen diet of thrice-boiled vegetables. Dr. Hansen thought her comatose patient at Northwestern Hospital deserved an opportunity to try insulin. Where to get it? She wondered if Dr. Robert Green, medical school bacteriology professor, might know, since his wife was a diabetic. Sure enough, Dr. Green was able to obtain some for her. Dr. Moses Barron says that Dr. Hansen's case was the first private patient in Minneapolis treated by insulin. She continued her interest in the use of insulin and wrote several papers for publication.

Writing papers was not a new experience for Dr. Hansen. She had done research in pharmacology as a student under Dr. Hirschfelder and published, among others, an article on "Toxic Effects of Arsenic" as well as one with Dr. Corbett on "Experimental Jamdice."

Mother Hansen, nee Sophie Thomsen, was born in 1861 in Denmark and came to the United States in 1882. She found room and board with a Danish family in Chicago. Little did she know that it was the home of a cousin of her future husband! Sophie was a competent seamstress and worked in homes as a dressmaker.

Ole Peter Hansen, born 1856 in another section of Denmark, came to Michigan with his parents and 4 brothers in 1872. After his father died in a lumbering accident, Ole Peter, his mother, and

brothers moved to Dakota Territory to stake a claim in Brown County. During the spring of 1887, Ole Peter returned to his birthplace to visit the girl chosen by his mother to be his mate. En route, he visited his cousin in Chicago, and there he met Sophie Thomsen. The romance was consummated in marriage June 1887, Ole Peter having stopped in Chicago on the return trip from Denmark and made arrangements with Sophie to come to Brown County for the ceremony. The couple established a homestead in Dakota Territory and managed to survive the pioneer hardships of blizzards, sandstorms, prairie fires, and drought. Two years later, in 1889, North and South Dakota were admitted to the Union as the thirty-eighth and thirty-ninth states.

Olga Sophie, first child of Sophie and Ole Peter Hansen, was born March 18, 1890, on her parents' "Jim River" farm near Hecla, northeast of Aberdeen, South Dakota. Her brother, George, born two years later, died in the 1918 influenza epidemic. Brother Frederick, five years younger, is a federal veterinarian in charge of Wichita, Kansas, stockyards. His son, Frederick, Jr., is federal veterinarian in charge of Oklahoma Disease Control.

Olga learned the Danish language from her paternal grandmother who, until her death at 84, lived in the Hansen home but never spoke English. Perhaps Olga had a Danish brogue, but it was not as evident as her cousin John's. As a child, he called her "cuss Olga." He is now Dr. J. G. Thomsen, dermatologist in Des Moines, having graduated from Iowa University Medical School.

Olga, at 8, entered a country school a mile away to which she walked each day. The teacher, who usually boarded at the Hansen home, went to school an hour earlier "to start the fires."

Mother Hansen was by far the most modern of housewives in the area. She was an excellent cook, was interested in physiology—used charts to show the function of the stomach—and was a member of the School Board. Another of her charts was a hob-nailed liver showing the ill effects of alcohol. She was a guiding light and president of the Women's Christian Temperance Union, and her children were members of the Loyal Temperance Union. Having signed the pledge never to swear, smoke, or drink, each child entered the summer declamatory contests decrying the evils of alcohol. Olga recited the *Face on the Bar Room Floor*, having practiced it in the haymow, and was awarded a silver medal.

At first, church services were held once a month in the schoolhouse. A circuit Lutheran minister from the county seat preached and each time was invited to the Hansen home for Sunday dinner. Later, a Union Sunday school was started in the town of Hecla. Mother Hansen taught in this interdenominational church school. She was open-minded, and her children benefited by her interest in new ideas.

Olga was 9 years old when father Hansen traded a cow for an organ. The school teacher gave Olga lessons, and father Hansen insisted that Olga play whenever the family had guests.

"We were constantly encouraged to read." A calf would be sold and more books purchased. Already in the library were the Bible and classics of Dickens and Shakespeare as well as magazines, such as *A Youth's Companion* and the *Christian Herald*. Both parents devoted their lives to education. The neighbors reared their children to be the "hired men" on their farms but not so the Hansens. Their motive was always education. Of course the Hansen children helped with the chores. Since Olga was the oldest, she was her father's "best hired man." She liked horseback riding and, at age 7, was put in charge of herding the sheep, coming home for lunch and to sleep but spending days in the saddle.

Other years she pitched hay, milked cows, and helped with the plowing. With no trees on their claim, a fire break was maintained by plowing a wide strip of furrows as a protection against prairie fires. During the frequent drought and depressions in South Dakota, many settlers abandoned their farms and moved to the cities. Father Hansen said, "We don't have enough money to leave," so he began to raise purebred, registered cattle and gradually accumulated land on sale for back taxes.

When 14, Olga was ill with severe tonsillitis followed by rheumatic fever that kept her out of school for one year. She then started high school at Aberdeen. By the next fall, the Hecla High School was completed with a total faculty of 2 teachers, who taught all the subjects offered. With 4 other classmates, Olga graduated in 1908.

Each summer was highlighted by the trip to see the "Greatest Show on Earth," the Ringling Brothers' Circus. Eggs and butter had been traded for groceries days in advance. Milking on the day they were to see the circus was started at 3 A.M., and, by 5 A.M., basket lunch and children were packed in the wagon, and the Hansen family began the 35-mile trek to Aberdeen. Dinner at noon in the Sherman Hotel was the special treat that preceded the circus. The basket lunch was reserved till after the performance. Then, on the return trip, each child, dead tired, slept all the way home, despite the biting mosquitoes.

After high school graduation, Olga attended Northern Normal and Industrial School, now Northern Teachers College, at Aberdeen. Meantime, her parents had sold their property in South Dakota and moved to Bird Island, Minnesota, so that their children would be closer to schools. The next year, 1910, with her certificate from normal school, Olga entered the University of Minnesota. One academic year plus one summer session and the credits from normal school permitted her to enter medical school in the fall of 1911.

About this time, the Hansens moved to the St. Anthony Park section of St. Paul. Many get-togethers of the women medical students were held at their home, with praises galore from the students of mother Hansen's wonderful cooking. Graduating from medical school in a class of 36, Olga was the only girl. As "top man" in the class, she was elected to the honor fraternity, Alpha Omega Alpha. Her

organization memberships and staff appointments make a long and imposing list:

Staff physician at St. Mary's and Northwestern hospitals, Minneapolis, since 1916. Served as secretary for the latter as well as physician to the Nurses Training School.

Clinical instructor of University Hospital outpatient department from 1917 to 1927.

Member of Hennepin County Medical Society and American Medical Association since 1917.

Staff physician at Doctors Memorial Hospital since 1930; chief of staff in 1953, reelected in 1954.

Charter member Board of Directors of Women's Auxiliary of Doctors Memorial and lifetime member of Executive Committee liaison to staff and auxiliary.

Charter member of the Minnesota Society of Internal Medicine.

Elected to American College of Physicians in 1927.

Certified as diplomate of American Board of Internal Medicine in 1928.

Consultant cardiologist at Glen Lake Sanatorium since 1928.

Member of American Heart Association.

Member of Minnesota Pathological Society.

Member of Diabetes Society.

Member of Minneapolis Society of Internal Medicine.

Grand chapter president of Alpha Epsilon Iota from 1936 to 1938, and longtime secretary of the alumnae section of the Minnesota (Epsilon) chapter.

Member of Unitarian Society.

Member of Faculty Women's Club.

Member of Zonta Club.

Lecturer, Minnesota Branch of American Pen Women.

Member of Women's Symphony Association.

Member of Latin American Club.

Member of Minneapolis Woman's Club.

Member of Lifetime Club, Arthur Murray School of Dancing.

This list gives some indication of Dr. Hansen's output of energy, the many facets of her personality, and her interests that extend beyond the practice of medicine. "I have had my full quota of memberships, committee appointments, and offices according to my ability. I believe my colleagues accept me as one of them. No one hushes his voice when I appear."

Many of her colleagues wish they had some of her boundless energy and good humor. "I'm just a show-off," says Dr. Hansen; "An extrovert," say her friends. Hundreds of graduates can say with pride, "She was *my* teacher."

Dr. Hansen has always believed that a woman who is a physician is a physician first and subject to the same motivation as a man who is a physician. Thus, she will be permitted to take her place in professional organizations on the same merits. This valuable philosophy has been an inspiration to class after class of women medical students to whom Dr. Hansen has been friend and advisor. To these same students she has made herself and her home available for counsel and for meetings.

Many times, Dr. Hansen has been asked such questions as, "Aren't you sorry you are a doctor instead of a lady?" or "Don't you lady doctors have a hard time in this man's world?" Dr. Hansen believes she has never been treated unfairly but neither has

she looked for special favors. She has felt confident working with men, finding no bias or discrimination. On the contrary, she says, "I have always had backers like Dr. S. Marx White, and it seems when one is in the minority, if at all possible, one gets the extras."

After a radical mastectomy in late May 1959, deep x-ray therapy followed through the month of June. During July, Dr. Hansen accompanied her brother Fred and his family on an automobile trip to the West Coast. By mid-August, she had returned to full-time practice at the Nicollet Clinic and had resumed her dancing lessons. This demonstration of

stamina is typical of Dr. Hansen's amazing energy and indomitable spirit.

The author wishes to thank Dr. J. Arthur Myers, chairman of THE JOURNAL-LANCET's Board of Editors, for the privilege of being asked to record Dr. Hansen's life in print—to be sure, a sketchy and partial expression of her ability and contributions to the world. The author first met Dr. Hansen in 1922 at the Nicollet Clinic while working as switchboard operator. She then worked under her as electrocardiographic technician, part-time student employee during academic years 1923 to 1926. Now, as a colleague since 1936, it is an honor and a pleasure to describe this integrated, complex, charming, and capable woman physician to your readers.

PHEOCHROMOCYTOMA, a rare medullary adrenal tumor, produces sustained or paroxysmal hypertension, often accompanied by hypermetabolism and an increase in blood sugar. Accurate diagnosis is essential, since surgical removal of the tumor may save the patient's life. No test is infallible, and the greatest obstacle to correct diagnosis is previous medication.

After the patient has been resting comfortably for at least one-half hour, blood pressure is determined in both arms. If a significant difference is noted between pressures in the two arms, pressure is determined simultaneously in both arms during the tests.

Unless the diastolic pressure is more than 150 mm. Hg, the cold pressor test is performed. Response of the pressure to cold will be compared with the reaction to histamine.

Following the cold pressor test, when the blood pressure has returned to basal levels, 0.05 mg. of histamine base in 0.5 cc. of normal saline is injected intravenously with a tuberculin syringe. While the needle remains in the vein, the empty syringe is replaced by one containing 5.0 mg. of Regitine.

Blood pressure determinations are made every thirty seconds for two minutes. A fall in pressure, always occurring thirty seconds after intravenous injection of histamine, stimulates secretion from the tumor. After the initial decrease, the pressure rises sharply and symptoms of a severe attack will be seen in a patient with pheochromocytoma. In such instances, the pressure increase will be greater than that induced by the cold stimulus.

Regitine is injected two minutes after injection of histamine, or sooner if necessary. Within one minute, blood pressure decreases as the attack subsides. Further administration of intravenous Regitine may be necessary if symptoms reappear due to the presence of a large or active tumor.

When basal blood pressure is greater than 170/110, the Regitine test is made. Intravenous administration of 5 mg. of Regitine will cause a 35/20 decrease in pressure during the first three or four minutes after injection if the test is positive; pressure will then return to basal levels within ten to fifteen minutes.

Sedatives or narcotics given within forty-eight hours preceding the test will cause false-positive results. Antihypertensive drugs should not be given for eight to ten days before testing.

Chemical tests may also be made with the use of pressor amines and catecholamines. Epinephrine and norepinephrine in the blood, determined by fluorometric methods, are increased in patients with sustained hypertension and during the height of a paroxysmal attack caused by pheochromocytoma. Blood should be collected during the histamine test at the pressure peak, since normal amounts of epinephrine and norepinephrine may be present in blood collected while the tumor is not secreting.

Among 504 patients without pheochromocytoma, the average concentration of urinary catecholamines was 114 mg. for twenty-four hours. In 4 patients with the tumor, the concentration varied from 42 to 493 mg. per 100 cc. thirty minutes after administration of histamine.

G. M. ROTH, E. V. FLOCK, J. L. BOLLMAN, and W. F. KVALE: Evaluation of the pharmacologic and chemical tests as an aid to diagnosis of pheochromocytoma. *Angiology* 10:426-430, 1959.

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BOOK REVIEWS

Year Book of General Surgery—1959-1960

MICHAEL D. DE BAKEY, M.D., *editor*, 1959. *Chicago: Year Book Publishers*. 620 pages. Illustrated. \$8.00.

This annual review of the important contributions to surgical literature is a valuable addition to every accomplished and aspiring surgeon's library. The volume is arranged in sections, with concise, meaty summaries of published papers along with the reference to the complete manuscript. Illustrations are generous and adequate, and techniques are amply described. Interspersed among the abstracts are pithy, pertinent comments by the editor—particularly in the area of cardiovascular surgery. As a commentary in the surgical literature of the past year, it is interesting to note that, of 525 pages devoted to the entire domain of surgery, 129 pages are devoted to the heart, aorta, and peripheral arteries. This is an accurate reflection of current surgical interests.

More or less as an addendum is a 100-page section on anesthesia edited by Dr. Stuart C. Cullen. Although interesting and informative, this might have been more advantageously incorporated or expanded into a separate volume.

ARNOLD J. KREMEN, M.D.
Minneapolis

Diseases of Metabolism

CARFIELD C. DUNCAN, M.D., *editor*, 1959. *Philadelphia: W. B. Saunders Co.* 1,101 pages. Illustrated. \$18.50.

This text has been a titleholder without a major contender for many years. Compared to itself in earlier editions, it is more sophisticated, as is the science of metabolism, and as effective in its attempt to bridge "the gap between the highly technical basic concepts and their clinical application." As more is learned about the body chemistry, the significant role of metabolism is more apparent. Improved understanding of normal and abnormal metabolism becomes imperative in actual practice.

The text provides no week-end refresher course in this wide field. Nevertheless, there seems to have been a concerted effort by editor and contributors to provide complex material in a highly readable form.

Virtually half the book is devoted to normal metabolism with effective sections on nutrition; absorption; digestion; metabolic pathways; and the fate of protein, carbohydrate, and lipid. This is followed by mineral metabolism, water balance, vitamins, and under- and overnutrition. Thereafter are sections on the special clinical disease states in metabolism. Some of these are uncommonly encountered (glycogen storage disease, porphyria, diabetes insipidus), while others make up a very important proportion of medical practice (mellitus, diabetes mellitus, diseases of the thyroid and the kidney). Knowledge of any of these complements knowledge of the others.

It is sad that we must still deal with appendix tables

of body build based on men and women in shoes, in clothes, and dressed in the manner of the latter 1800's.

The volume is otherwise remarkably up-to-date and should effectively aid the physician's understanding, appreciation, and care in dealing with new hypoglycemic drugs, cholesterol depressing agents, active thyroid substances, and the like. It is a valuable reference for the student, and the bibliographies of each section are more valuable than those found in many texts.

It is a scholarly volume yet consciously concerned with ready availability of knowledge to the reader seeking a reference and with practical application in diagnosis and therapy. It would appear that this edition might be used with benefit by readers of any level of understanding in the field of metabolism.

HENRY W. BLACKBURN, M.D.
Minneapolis

Some Papers on the Cerebral Cortex

GERHARDT VON BONIN, *translator*, 1960. *Springfield, Ill.: Charles C Thomas*. 396 pages. Illustrated. \$11.50.

Gerhardt von Bonin, a distinguished professor of neuroanatomy, has been too modest in his choice of a title for this book. This volume certainly deals with the cerebral cortex, but, in a larger sense, it traces the historic patterns which have led to most of our modern concepts about the function and structure of the cerebral hemispheres and the central nervous system.

Dr. Von Bonin has grouped 12 essays on the cerebral cortex in an extremely logical fashion. Of these, 11 were written originally in either French or German. His lucid and accurate translation of the original text is in itself a reason for reading the book.

The essayists include Pierre Flourens, Jules Gabriel Francois Baillarger, Paul Broca, Fritsch and Hitzig, and Hermann Munk, who describe their pioneering work on the function and structure of the cerebral cortex. Friedrich Goltz gives his classic paper on cerebral hemispherectomy in the dog, and Theodor Meynert expresses some very sophisticated ideas about the collaboration of parts of the brain in producing their total function. Paul Flechsig writes a spirited paper on brain physiology and the theories of volition. Korbinian Brodmann presents his work on histologic localization of the various parts of the cerebral cortex, and von Monakow delivers an elegant essay that deals with the integrated function of the cerebral hemispheres and the effect of diastasis on brain function.

The last 2 essays are of particular interest. Ramon y Cajal is usually thought of in conjunction with his remarkable studies of cellular histology and pathology. The essay presented in this book deals with his thoughts on the function of the cerebral hemispheres and includes some sharp criticisms of the anatomic and physiologic studies preceding his investigations. His reserve about drawing definitive conclusions on the function of the

(Continued on page 24A)

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BOOK REVIEWS

(Continued from page 22A)

brain in the absence of sufficient anatomic and physiologic evidence is commendable. Leyton and Sherrington have written the only English essay to be reproduced in this volume. Their observations on the excitable cortex of the chimpanzee, orangutan, and gorilla is a fundamental contribution to cerebral physiology.

Dr. von Bonin, in his introduction, gives a lucid criticism of each of the essays presented in his book as well as a little historic résumé of the individual who wrote the essay.

The book is clearly printed and appropriate illustrations are included with the essays.

Although the average reader may find this book a bit too detailed, students of the nervous system, be they clinicians or basic scientists, will find this monograph a valuable addition to their library.

VERNON H. MARK, M.D.
Boston

The Pharmacology and Clinical Use of Diuretics

CARROLL A. HANDLEY, PH.D., and JOHN H. MOYER, M.D.,
1959. Springfield, Ill.: Charles C Thomas. 162 pages.
\$6.00.

A decade ago, any discussion of diuretic agents automatically limited itself to mercurials, xanthenes, and urine acidifiers, and a few simple rules covered their clinical use. However, with the development of such agents as Diamox and Diuril, the diuretic field appears to have suddenly become complex, for an ever-expanding therapeutic usefulness of these agents seems to outstrip a complete understanding of their physiologic actions. The authors of *The Pharmacology and Clinical Use of Diuretics*, clearly see this problem and point their discussion toward the practicing clinician, be he general practitioner or cardiologist, providing him with an up-to-the-minute manual which he will want to keep close by for ready use. This book is not an attempt at oversimplification of a complex subject but is a clear statement of principles governing the use of all diuretics, graphically illustrated and backed by well-documented clinical experience. Their style of writing is so pleasing and the length of the book such that one can enjoy reading it through in one evening. Large print and uncluttered demonstration figures help to keep the learning process from becoming a chore, and yet the reader is left with the distinct feeling that the subject has been completely covered.

Chapters 3 to 11 cover organomercurials, carbonic-anhydrase inhibitors, chlorothiazide, xanthenes, triazines, acidifying agents, and antialdosterone drugs—their actions and their side effects. The authors write in building block form, comparing the action and effectiveness of these drugs with those previously described; in this way, the clinician's basic knowledge of standard diuretics is used as a foundation from which to build an understanding of the recently developed agents. Chapters 1 and 2 produce valuable digressions into the mechanisms involved with salt and water control in the normal person and the patient with cardiac failure. To one deeply interested in fluid-electrolyte balance, these chapters alone are worth the price of the book, for the authors dare to state logical principles involved in electrolyte balance without the usual confusing qualifications that keep this phase of medicine such a frightening one to the clinician. Chapter 11 presents the working summary of diuretics in the various edema states, giving indica-

(Continued on page 26A)

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BOOK REVIEWS

(Continued from page 24A)

tions for and rules of use. These last four pages will probably become most thumb-worn on the physician's desk.

This book is recommended to the conscientious clinician and medical student as the most complete and concise working manual on diuretics yet written.

RICHARD P. LYON, M.D.
San Francisco

The Surgical Treatment of Facial Injuries

VARAZIAD HOVHANNES KAZANJIAN, M.D., and JOHN MARQUIS CONVERSE, M.D., 1959. *Second edition*. Baltimore: Williams & Wilkins Co. 1117 pages. Illustrated. \$22.00.

This is the second edition, completely revised, considerably enlarged, and updated, of a textbook which has for ten years maintained its position as a real contribution to surgery in general and to the specialties of plastic, maxillofacial, ophthalmic plastic, and oral surgery.

The text designed for the student, practitioner, and specialist comprises fifty years' experience of the senior author, a pioneer and a remaining leader in plastic surgery, and twenty years' collaboration and experience of the junior author, whose own contributions are well acknowledged in the specialty. Contributing authors of various sections, both revised and new, are recognized authorities.

Six new chapters and 400 new illustrations have been added. The drawings are excellent and serve admirably

to illustrate significant features of the problems discussed. The text is well constructed, nicely organized, and pleasant to read.

The first 3 chapters discuss the evolution of the human face, some aspects of anatomy, healing of wounds, and general principles of operating technique. Significant present-day concepts are clearly presented. The chapter on "Early Treatment of Facial Injuries" should be studied thoroughly by all physicians dealing with trauma. The results of "average" treatment of such injuries would be improved measurably by application of the principles presented. The chapters dealing with fractures of the bones of the face and jaws are classic. Although the many dental apparatuses discussed are not now commonly employed, principles of treatment are emphasized by their application. The discussion of the etiology and treatment of diplopia and exophthalmos associated with fractures of the zygoma is a particularly significant contribution because of the average lack of understanding of this problem. The new chapters on "Fractures of the Fronto-Ethmoidal Region," "Facial Injuries in Children," and "Scars of the Face" are significant additions to the text.

The latter half of the book is devoted to general principles of reconstructive surgery of the face. The material is well illustrated both by diagrams and case presentations, and the discussions are remarkably complete. This portion of the text will serve admirably for reference study.

The book is recommended to all physicians confronted with the problems of trauma and to specialists in reconstructive surgery.

JOHN D. DESPREZ, M.D.
Cleveland



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Current Surgical Procedures for Carcinoma of the Rectum

EDWARD S. JUDD, M.D.

Rochester, Minnesota

THE PRESENT-DAY concept of the proper treatment of cancer of the rectum has been predicated on the work of Sir William Ernest Miles, who first clearly emphasized the modes of spread of this lesion in 1908. It is now well established that these tumors spread in 4 general ways: (1) through the regional lymphatic vessels, (2) through the veins, (3) by direct extension of the lesion, and (4) by implantation on the peritoneal surface. Miles was the first to be convinced that the spread could be downward and lateral as well as upward. For that reason, complete sacrifice of the anus and its sphincters appeared necessary in every case. In the light of present-day knowledge, however, some of the lesions situated at a somewhat higher level can be treated without such radical means. Any operation for carcinoma must entail complete removal of all the routes of metastasis if a cure is to be realized. Proper judgment in selection of the proper operation for each patient may mean that occasionally the anus and the anal sphincters can be preserved.

EDWARD S. JUDD is head of a Section of Surgery at the Mayo Clinic and professor of surgery at the Mayo Foundation of the University of Minnesota Graduate School of Medicine.

Paper presented at the meeting of the North Dakota State Medical Association, Grand Forks, April 30 to May 3, 1960.

METASTASIS VIA REGIONAL LYMPHATIC VESSELS

This method of spread depends entirely upon the location of the cancer. If the lesion is situated in the lower third part of the rectum, the metastatic route is largely in an upward direction toward the lymph nodes located around the superior hemorrhoidal vessels. However, it is entirely possible that spread also may take place along the levator ani muscles and even into the ischiorectal fossa. There can be no doubt that any curative procedure performed in such a situation must include removal of the levator ani muscles, ischiorectal fat, all the skin surrounding the anus, and the entire rectum, sigmoid, and mesosigmoid. It is obvious that such an operation demands a permanent colonic stoma.

If the carcinoma is located above the level of the lower third part of the rectum, metastasis by way of the lymphatic vessels still proceeds upward along the superior hemorrhoidal system. However, in this instance, the spread caudally and laterally develops only in advanced carcinoma, when those more superiorly located lymphatic vessels already have been blocked off by malignant invasion. It appears, then, that the surgeon may consider preservation of the sphincter mechanism in the presence of certain types of carcinoma of the middle and upper parts of the rectum. However, should the tumor be extremely anaplastic or unusually bulky and exten-

sive, sacrifice of the entire rectum still may be required for these reasons alone.

LOCATION OF LESION

Considerable confusion arises in respect to location, for there is no standard method of expressing or denoting the exact level. In general, a tumor of the lower part of the rectum is very easily palpated in its entirety by routine digital examination of the rectum. Many lesions situated at a higher level can be at least identified by the examining finger, especially if the patient co-operates in straining forcibly during the examination in such a manner as to prolapse the lesion. Proctoscopic examination is an indispensable factor in this diagnosis. It is quite a convenient way to measure the distance between the lower border of the cancer and the anus. Care must be taken to be consistent in stating whether the anus itself or the so-called dentate margin of the mucocutaneous junction is the determining mark. As long as consistency is maintained in this respect, there should be no confusion. It is realized that many of these tumors are intussuscepting and prolapsing, and thus proctoscopic measurement alone is not the final criterion. However, in general, lesions located between the dentate line and up to the 5-cm. level may be thought of as being in the lower third part of the rectum; those 5 to 10 cm. between these points are in the midrectum; and those located more than 10 cm. from the dentate line are considered by my colleagues and me to be situated in the upper part of rectum or rectosigmoid.

The fallacy of reliance upon a roentgenogram of the colon cannot be overemphasized in this discussion because, from the standpoint of roentgenography, the rectum and rectosigmoid are "blind spots." The experienced roentgenologist will report the colon in his study exactly as he sees it, but he will also emphasize the fact that he is able to delineate the colon only beyond that level easily encompassed in proctoscopic examination.

PREPARATION OF PATIENT

First and foremost, the surgeon must approach the patient with as sympathetic an attitude as possible, keeping the psychologic factors uppermost in mind at the original interview. He will point out that occasionally the only possible hope of cure lies in radical sacrifice of the entire rectum, leaving the patient with a permanent colonic stoma. In some instances, he will also point out that a less mutilating operation may be equally curative if the patient is fortunate

enough to have a lesion situated at a high enough level. At the Mayo Clinic, we consider combined abdominoperineal resection the procedure of choice in most cases, and we are always prepared to proceed with it in any questionable situation. It is a common experience for a patient who is badly agitated at the thought of a colonic stoma, and who places perhaps undue significance upon it at the time of the original interview, to adjust to the new situation very quickly, so that he has already learned to accept it as a matter of course by the time he reports for the six-month postoperative examination.

Physical preparation of the patient has become most effective. It goes without saying that anemia, malnutrition, general metabolic disorders, and other derangements now can be corrected quite fully and quickly if their existence is known in advance. The alert surgeon will have at his command a host of adjuncts and measures to transform the patient from one who represents a desperate risk into one who constitutes a much more acceptable risk. Usually, there is adequate time in which to delay operation for a few days if there are specific factors which may be overcome by these modern measures.

Preparation of the colon and rectum is most essential if morbidity and mortality rates are to be kept at an acceptable level. We have insisted on a three-way project, namely, rather forceful purging of the bowel, followed by an extensive irrigation program and a sterilization routine with antibiotic agents. At present, we require at least two full days in the hospital for this preparation to be carried out properly. The antibiotic agent employed varies according to the wishes of the surgeon, but, on my own surgical service, we have been using neomycin for the past several years with excellent results. We are not able to render the colon totally sterile with such a preparation, but thus far we are happily free of many of the problems involved with staphylococci which might ensue if a mixture of the other antibiotic agents were employed.

CHOICE OF OPERATIVE PROCEDURE

We have made it a rigid rule that our initial abdominal dissection will be the same regardless of the type of operation to be performed. I prefer a low left rectus incision which extends all the way to the periosteum of the pubis. The liver is examined first to determine whether there is any palpable implant. Since we usually avoid a roentgenogram of the colon in any case in which obstruction by the lesion might com-

plicate the situation after the introduction of barium, we palpate the colon very carefully, because it is not very unusual to detect a separate carcinoma in the proximal portion of bowel. When the operability of the tumor has been determined, we proceed with wide incision of the peritoneal leaflets of the mesosigmoid, elevating these flaps extensively and as far away from the bowel as we can. The spermatic (or ovarian) vessels are isolated immediately, and the position of both ureters is determined at once. Generally, the ureters are exposed extensively and packed out of the way, care being taken not to compromise the blood supply to these structures. The rectosigmoid and rectum are freed to the level of the levator ani muscles, which means that the coccyx is freely palpable from above. The mesentery is divided at the aorta, so that all the lymphatic vessels are secured at a high level. The middle hemorrhoidal vessels are divided as far laterally in the pelvis as the surgeon can reach, care being taken once again not to damage the ureters. The anterior attachments are severed directly away from the base of the bladder and the prostate or vaginal wall, as the case may be. It is at this point that the decision is made as to whether the patient must undergo combined abdominoperineal resection or whether the extensive mobilization just described has brought the tumor well up into the pelvis so that a good length of normal bowel below the site of the tumor can be included in the resection, permitting preservation of the lowermost part of the rectum or, at least, the sphincters. It is well to ligate the bowel above and below the site of the tumor to prevent dissemination of malignant cells within the lumen.

OPERATION

Combined abdominoperineal resection. If the tumor is found to be situated so low in the bowel that it is not possible to divide that structure at least 4 cm. below it, we abandon all thought of a sphincter-saving operation and proceed with the classic abdominoperineal resection of Miles. The descending colon or uppermost part of the sigmoid is divided and brought out through a lateral stab wound to serve as a permanent single-barrel colonic stoma. The other end of the sigmoid is closed by suture. The bowel is then folded over and tucked into the huge presacral cavity which has been produced by dissection. In obese patients with a very narrow pelvis, it may be necessary to amputate a portion of the sigmoid to accommodate the remainder in this cavity. The new pelvic floor is then constituted

by suturing the eut peritoneal borders together. This may require rather extensive elevation of the lateral flaps of peritoneum and may call for some ingenuity in effecting complete closure. In a female, the new floor may be fortified by employing the uterus, tubes, and ovaries, if still present, for such a purpose.

After closure of the abdominal wall, the patient is placed in the lithotomy position. A wide perineal incision is made, encompassing much of the perianal skin. All this tissue is then removed, dissection proceeding directly from the bony pelvic walls. Posteriorly, the coccyx can be removed quite easily and the muscles are divided as far from the rectum as possible. Anteriorly, dissection proceeds directly against the vaginal wall or the prostate gland and membranous urethra, as the case may be. After hemostasis has been secured, the wound can be partly closed with widely spaced mattress sutures, and a surprising degree of approximation can be realized. Drains can be inserted in the remaining space, although I still prefer to insert a gauze pack, because a large cavity remains which can be healed only by means of granulation tissue.

Low anterior resection. If, at the completion of the mobilization described, it appears possible to divide across the rectum at a safe distance below the lowermost border of the tumor—preferably 5 cm. or more—the surgeon can proceed, with preservation of the lowermost part of the rectum, so that end-to-end anastomosis can be effected between the uppermost part of the sigmoid and the rectum. This eliminates the need for colostomy and preserves normal fecal control. Earlier, certain authorities discouraged the use of this operation. However, it is now apparent from our own records that, when properly selected, the operation has an excellent application and is associated with satisfactory morbidity and mortality rates.

The change in thinking has come about, first, because we now realize that the spread downward or laterally, by way of the lymphatic vessels, is minimal when curable carcinoma of the rectum is located above the levator ani muscles and, second, because modern adjuncts to surgical management have greatly reduced operative mortality rates. We now use this operation frequently when dealing with cancers of the rectosigmoid and even with some carcinomas located in the upper, and possibly the middle, third portion of the rectum. A slender woman with a wide pelvis will present a much better opportunity for successful completion of the operation than will an obese man with a very

narrow pelvis. Several of our surgeons insert a rectal tube directly through the anastomosis and anchor it in position for five or six days. Others simply dilate the anus. Currently, it is much less common to employ a temporary proximal colonic stoma, although we do not hesitate to perform this operation if we are not satisfied with the appearance of the anastomosis. The lower segment of bowel is completely devoid of any serosa, and healing may be slow. We always insert drains, bringing them out a separate incision. Fecal leakage may occur, and we try to be prepared for it. Ordinarily, it can be controlled without great difficulty.

Pull-through operation. After complete mobilization of the rectum, it may be apparent that the tumor has been elevated from its original position to a point at which the surgeon is reluctant to remove the entire anus. If it appears that there is an adequate portion of normal rectum below the site of the lesion, resection and performance of end-to-end anastomosis still might be technically impossible. In such a situation, resection may be carried out; instead of performance of end-to-end anastomosis, the uppermost part of the sigmoid may be drawn down through either the bared anal sphincter or the intact anal mucosa in a deliberate intussuscepting type of union. This method is particularly applicable for those lesions situated between 5 and 10 cm. from the anus, whereas low anterior resection very frequently can be performed to eradicate those lesions more than 10 cm. from the anus. Pull-through operations must never be used instead of the combined abdominoperineal resection but occasionally may be used instead of low anterior resection. The indications may be somewhat limited, but surgeons who are practiced in performing the pull-through operation are enthusiastic about it. Sometimes, fecal control is not ideal after a pull-through operation, and many patients thus treated wear a pad for security purposes. It is said that, if the type of pull-through operation which employs the intact anal mucosa is performed, the patient will have more of a warning that stool is gathering proximal to the anus, whereas, if a procedure is done in which all the mucosa is stripped away, the proprioceptive fibers governing the sensation of fullness will have been lost.

POSTOPERATIVE COMPLICATIONS

Vesical dysfunction is common among many of these patients; it is perhaps the most frequent sequel of combined abdominoperineal resection. In a male, transurethral prostatic resection may

well be required, especially in view of the fact that most of the patients are elderly. Infection of wounds still occurs, even in this era of thorough intestinal preparation. Carcinoma of the rectum frequently is perforating and, even though great caution has been exercised, infection occasionally may develop. Since the patients concerned are, for the most part, older, cardiovascular complications are not rare. Pelvic abscesses after sphincter-saving operations usually can be prevented by the establishment of adequate drainage. Formation of stricture at the site of anastomosis is not common after anterior resection and, although it occasionally follows pull-through operations, it can be overcome by digital dilatation.

CARE OF COLONIC STOMA

In the case of the temporary colonic stoma, still used in isolated instances in association with low anterior resection, we have not gone into great detail with the patient in respect to care and maintenance because we usually close the colonic stoma at a very early date. Several types of colostomy bags now available are very satisfactory and have the feature of disposable receptacles. The patient quickly learns how to plan his diet so that he can manage his colonic stoma for a short time.

The patient learning how to care for a permanent colonic stoma requires more careful supervision and training. We have supplied the patient with a booklet of our own design which outlines diet, proper method of irrigation of the stoma, use of colostomy bags and appliances, and necessity for digital dilatation. Ordinarily, we prefer to leave enough of the sigmoid protruding above the level of the skin so that a bag can be worn easily without spilling. Some people prefer to use only an elastic garment to hold "cellucotton" or tissue in place and do not require or even want a bag.

The problem of irrigation is an individual matter. We ask the patient to start irrigating the stoma three times a week after he returns home. He then learns how much water to employ, what time of day to irrigate, and whether he prefers to irrigate more or less frequently. We have learned not to be rigid in our counsel on this problem. Some patients discontinue irrigation entirely and maintain themselves very well without it. Others irrigate four or five times a week and have no discharge of stool at any other time. These seem to be the happiest patients, and they include those who have dispensed with the bag. Patients quickly learn which foods produce diar-

rhea and which produce constipation, and they then balance their diets easily between the two categories.

We are prepared for a patient to be somewhat disturbed and nervous for perhaps six weeks, but we are gratified to note how rapidly a sense of security develops. Occasionally, a patient gains much weight, and the fat tends to close down over the stoma, so that the scar at the edge then obstructs the bowel. As a rule, this complication can be overcome by reducing his weight and resorting to daily digital dilatation of the stoma. Once in a while, a minor plastic operation on the stoma is necessary to revise it. Paregoric and other medications are rarely required once the patient becomes accustomed to his stoma. Preparations formerly available for deodorizing the contents of the bowel have been largely abandoned.

SURVIVAL RATES

In the case of combined abdominoperineal resection, we found that, among all patients who underwent resection, with the exclusion of those with known metastasis to the liver, a five-year survival rate of 50 per cent was the rule. In a study¹ published in 1959, we found that the five-year survival rate was 67 per cent when the lymph nodes of the mesentery were free of disease. However, when the lymph nodes were involved, the five-year survival rate decreased to 24 per cent.

When low anterior resection was carried out largely for lesions situated in the upper third portion of the rectum and rectosigmoid, the five-year survival rate was 61 per cent. When none of the lymph nodes was involved, the rate was

76 per cent; whereas, when lymph nodes were involved, the rate was 44 per cent.

In the case of pull-through operations, we found that the five-year survival rate for all patients was 53 per cent. When lymph nodes were not involved, the five-year survival rate increased to 73 per cent; but, when the lymph nodes were involved, the five-year survival rate decreased to 28 per cent.

It appears that, as has been suggested repeatedly in the past, very low-lying rectal lesions are indeed associated with a somewhat poorer prognosis than are lesions at a higher level. It also appears that, even when such a lethal disease as cancer of the rectum is at hand, prompt and thorough surgical extirpation may well bring about a situation which is far from hopeless.

CONCLUSIONS

1. Combined abdominoperineal resection with creation of a permanent colonic stoma is still the operation of choice for a large share of cancers of the rectum.

2. Careful scrutiny of the pathologic process involved reveals that certain cancers of the mid-rectum and most cancers of the upper third part of the rectum can be handled satisfactorily without establishment of a permanent colonic stoma.

3. Accurate knowledge of the mode of spread of cancers in this region, combined with proper selection of the patients, and the present-day knowledge concerning adjuncts to surgical care have resulted in a more satisfactory outcome than was ever possible before.

REFERENCE

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DOGS WITH TWO-THIRDS of the small intestine removed can survive and eat ordinary food with no ill effects, but resection of 75 per cent or more causes diarrhea and death from starvation within three months. However, dogs with 80 to 90 per cent of small bowel excised can survive and maintain weight if 1 to 2 in. of the distal segment of remaining bowel is reversed and reinserted into the intestine to function in an antiperistaltic manner. The procedure might be applicable in cases of human mesenteric thrombosis.

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Brain Tumors

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TUMORS THAT ARISE in the intracranial cavity are many and varied. It has been estimated that brain tumors comprise between 1 to 2 per cent of all autopsies. Because many of these lesions are now operable if diagnosed early, every physician should have some knowledge of the characteristics of the different tumor types, their common locations, age and sex incidence, and growth characteristics. Since the different types tend to predominate in the different age groups, a discussion of their special clinical characteristics according to age groups will tend to simplify their diagnosis. There will be, of course, some overlap, since a number of tumors occur in more than one age group. However, for simplicity, only the more common types in each age group will be considered.

The symptomatology produced by brain tumors is governed by their histology and location. Rapidly growing lesions and those located in the motor cortex or along the ventricular system manifest themselves sooner than larger and more slowly growing tumors in relatively silent areas. Aside from the focal symptoms of brain damage, the most common general symptoms consist of persistent headache, vomiting, personality changes, convulsions, and papilledema. The gradual progression of any of these complaints with or without focal symptoms should always suggest the possibility of a brain tumor.

Intracranial tumors, although numerous, can be classified into large groups. A listing of these groups may be helpful in understanding the tumors to be discussed.

- I. Tumors of cranial nerves
acoustic neuroma
- II. Tumors of meninges
meningioma
- III. Tumors of brain parenchyma (gliomas)
astrocytoma, ependymoma, medulloblastoma, pinealoma, and glioblastoma
- IV. Mixed tumors
epidermoid

- V. Pituitary tumors
pituitary adenoma and craniopharyngioma
- VI. Metastatic tumors
lung, breast, and gastrointestinal tract
- VII. Vascular tumors
angioma

INFANCY AND CHILDHOOD, BIRTH TO 16 YEARS

Contrary to general belief, brain tumors are relatively common in this age group. It has been estimated that between 15 to 20 per cent of all brain tumors occur in children and that 1 in every 1,000 patients admitted to a general pediatrics clinic will have a brain tumor. Interestingly enough, the majority of tumors in this age group occur in the posterior fossa and involve the cerebellum.

Cerebellar astrocytoma. This is the most common tumor of children, comprising 35 per cent of tumors in this age group. It occurs in both a cystic and noncystic form. It is a slowly growing tumor that may be present for years, producing a dull headache as its only manifestation. As the lesion enlarges, the child may show some unilateral incoordination in the use of the limbs. Choked disk may be a late finding but usually is present in mild form when incoordination appears. In any child who complains of *headache and incoordination over a period of months*, an astrocytoma should be suspected.

Treatment consists of surgical removal. Since these are well circumscribed lesions, the prognosis is good. Cystic lesions can invariably be cured by early, adequate, surgical procedures.

Cerebellar ependymoma. This tumor arises from the subependymal tissues on the floor of the fourth ventricle and usually grows slowly to fill the ventricle. Because of its growth within the ventricle, symptoms do not occur until the tumor fills the entire ventricle and causes obstruction to the flow of spinal fluid. At that time, acute symptoms appear and consist almost exclusively of *acute onset of increased intracranial pressure with little or no cerebellar complaints*. The appearance of such a symptom complex in a child should always suggest a diagnosis of ependymoma.

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Surgical treatment is indicated and is usually palliative because of the large size of the lesion before symptoms appear. Certain tumors are moderately radiosensitive.

Medulloblastoma. This tumor arises from the roof of the fourth ventricle and is rapidly invasive, producing midline cerebellar symptoms. It is an extremely friable lesion that may scatter throughout the ventricles or spinal canal, thus giving rise to root pain or cord compression. It chiefly affects boys between ages of 4 to 8 years. This tumor type should always be considered in male children who develop *rapid onset of increased intracranial pressure* associated with a *gait disturbance due to trunkal ataxia*.

Treatment consists of surgical decompression for the increased intracranial pressure and deep x-ray therapy. These lesions are moderately radiosensitive, and good, though transient, response may be obtained from deep x-ray therapy.

Craniopharyngioma. This tumor is suprasellar in location and, when small, is limited to the region overlying the optic chiasm. As it enlarges, it compresses the optic chiasm and extends up into the third ventricle, destroying the hypothalamus.

The most frequent initial symptom is headache, often associated with vomiting. Compression of the visual system results in decreased visual acuity and optic atrophy. Hypothalamic involvement may result in somnolence, diabetes insipidus, Fröhlich's syndrome, and decreased glucose tolerance. These tumors often contain sufficient calcium to be visible on roentgenograms. Any child who complains of persistent headache and *decreased visual acuity* and has *bilateral optic atrophy* should be suspected of having craniopharyngioma. The diagnosis is verified by suprasellar calcification.

Treatment consists of surgically emptying the tumor contents. These lesions, although benign, cannot be removed completely because of their location.

Pinealoma. This tumor comprises only 2 per cent of tumors in this age group and occurs chiefly in males. As the tumor grows, it compresses the midbrain, resulting in the fairly rapid onset of signs of increased intracranial pressure. The most characteristic findings consist of ptosis and an *inability to turn the eyes upward*. Other oculomotor palsies may be present. Less commonly, bilateral hyperacusis or deafness and precocious sexual development may occur. Occasionally, a skull roentgenogram will reveal an enlarged calcified pineal gland.

Surgical removal of these tumors is difficult.

Treatment of choice is deep x-ray therapy preceded by an operation to shunt the cerebrospinal fluid around the blocked aqueduct and thus relieve the increased pressure.

Brain stem glioma. This group of tumors usually grows slowly and consists chiefly of astrocytic gliomas. Mild cranial nerve palsies, such as vertigo, diplopia, and facial pain, develop. These symptoms tend to slowly increase in intensity and are accompanied by long tract involvement, such as hyperactive deep reflexes, positive toe signs, or sensory changes. Bulbar symptoms may ultimately develop with respiratory and even cardiac symptomatology. The *slowly progressive development of cranial nerve involvement* usually confirms the diagnosis in this type of tumor.

Treatment is not available. Deep x-ray therapy may be tried but is usually without benefit.

ADULT TUMORS, 16 TO 45 YEARS

Cerebral astrocytoma. This is a slowly growing tumor of the parenchyma of the brain. It infiltrates throughout the brain tissue and produces symptoms of progressive brain dysfunction over a period of years. The signs and symptoms depend upon the region of the brain involved. Since this tumor occurs chiefly in the frontal and temporal regions, the patient usually shows evidence of slowly progressive motor weakness or mental changes with memory defects and confusional states. Because of its slow growth, evidence of increased intracranial pressure is often absent. The diagnosis should always be suspected in any patient with *progressive focal involvement over a period of years*. Verification of the diagnosis is carried out by special studies, such as angiography or air encephalography.

Because of their infiltrative nature, these lesions cannot be completely removed by surgery. However, the patient can be given palliative relief which may last for years.

Meningioma. This is a slowly growing tumor of the meninges which may become calcified and often produces hyperostosis of the overlying bone. Since these tumors are situated either on the surface or along the base of the brain, they frequently produce seizures or involvement of the cranial nerves. Meningeal tumors, when situated in certain localities, characteristically produce special diagnostic findings:

1. *Superior sagittal meningioma.* In its earlier stages, this tumor produces *focal seizures involving the leg*. This is followed after months by *slowly progressive weakness* of the involved leg.

2. *Olfactory groove meningioma*. Initially, there is *unilateral anosmia* followed by *personality or mental changes* due to involvement of the frontal lobe. As the tumor enlarges, it may extend posteriorly to implicate the optic nerves and produce *bilateral optic atrophy* and visual loss. Seizures, if they occur, are generalized.

3. *Sphenoid ridge meningioma*. This tumor slowly involves the orbit and the adjacent optic and oculomotor nerves. The presenting complaints are, therefore, *ipsilateral orbital pain* and a slowly progressive *ipsilateral exophthalmos*. These symptoms are associated with a *unilateral visual loss* and *optic atrophy* and, less commonly, an *ipsilateral ophthalmoplegia*. Roentgenographic evidence of bone erosion or bone production in this region is conclusive proof of the diagnosis.

Treatment of the meningiomas is most satisfactory. These are usually benign, well circumscribed lesions that can be completely removed by surgery with gratifying results.

Acoustic neuroma. This is a well encapsulated tumor arising from the vestibular portion of the eighth cranial nerve at the internal meatus. It often erodes the tip of the temporal bone and, as it enlarges, it compresses the lateral margin of the pons and medulla. Because of its origin, the earliest symptoms consist of *unilateral tinnitus* and a *progressive deafness*. Vertigo is less characteristic and may be mild or persistent. As the brain stem is compressed, corneal hypesthesia, facial palsy, and *ipsilateral ataxia* with gait disturbance develop. X-ray examination will reveal enlargement of the internal auditory meatus in one-third of the cases, and the spinal fluid protein is usually elevated.

Surgical removal results in a complete cure, particularly if the diagnosis is made while the tumor is still small.

Pituitary adenoma. This lesion always arises from the anterior lobe of the pituitary, and, as it grows, it first compresses the pituitary gland. In this stage, the tumor is often silent except for intermittent and often intense headache. Later, there is an enlargement of the sella turcica and erosion of the clinoids, which are readily apparent on roentgenograms. As the tumor extends outside the sella, it compresses the optic chiasm, producing a *bitemporal field defect* often associated with a *bilateral optic atrophy*. In very large lesions, the hypothalamus may be involved, producing adiposity, somnolence, and polydipsia.

Surgery is imperative when vision is failing or

if signs of increased intracranial pressure appear. Because of its location, it cannot be totally removed. If vision is still intact, irradiation may be tried with some success. In many cases, growth can be arrested indefinitely.

Angioma. This is a malformation rather than a true tumor. It consists of a plexus of abnormal vessels of varying size situated within the brain and often extending to the meninges. Clinical manifestations may appear early in life and continue for years, or symptoms may be delayed in onset. The most characteristic features are *recurring episodes of seizures* or *transient attacks of weakness* or other focal abnormalities often associated with subarachnoid bleeding. Occasionally, a cranial bruit may be heard. Some angiomas calcify and can be seen on roentgenogram.

Treatment depends upon the size and location of the lesion. A complete resection of the tumor is occasionally possible.

TUMORS OF MIDDLE AND OLD AGE

Many of the tumors of the previous age group also occur in older individuals. In addition, however, two other tumor types are seen with increasing frequency and should always be considered.

Metastatic tumor. This tumor comprises over 20 per cent of all intracranial neoplasms. It should always be considered in a patient in whom symptoms of an intracranial tumor develop rapidly in middle or later life, particularly if associated with *weight loss*, *pulmonary symptoms*, *radicular pain*, or *meningeal signs* without spinal fluid pleocytosis. These patients should be checked carefully for a primary lesion elsewhere in the body, paying special attention to the lungs, breast, gastrointestinal tract, and skin. In spite of a fairly rapid course, papilledema is often absent even in terminal cases.

Treatment is symptomatic. The course is usually rapidly downhill. Occasionally, surgery may offer palliative relief by prolonging life and making the patient more comfortable.

Glioblastoma multiforme. This malignant tumor of the brain parenchyma produces *rapid, progressive focal symptomatology* associated with *increased intracranial pressure*. The patient does not show a weight loss, and there is no evidence of a primary lesion.

Treatment consists of partial removal of the tumor. Life expectancy, even with surgery, is usually six to nine months.

Neuro-ophthalmology for the Busy Practitioner

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As 65 PER CENT of all intracranial disease causes ocular symptoms and signs, the importance of the neuro-ophthalmologic examination as a part of every physical examination can be readily appreciated. Time is not a great factor, for a fairly complete neuro-ophthalmologic survey can be made in about four minutes.

The walls of the orbit bound a combination of anatomic structures which, despite the complexity of their array, can be easily and precisely evaluated. Included are the second, third, fourth, and sixth cranial nerves; the ophthalmic division of the fifth cranial nerve; and the orbicular portion of the seventh cranial nerve. Nystagmus of specific types points variously to lesions of the labyrinths, of the vestibular nerves, of the pons, or of the cerebellum. Conjugate deviation of the eyes or paresis of conjugate lateral or vertical gaze may localize the responsible lesion in the cerebrum, midbrain, or pons. Disorders affecting the sympathetic system in its course from the hypothalamus to the cervical cord and upward again from the superior cervical ganglion to the eye may be encountered, as may lesions of the parasympathetic system to the eye. Lesions situated on the sphenoid ridge; in the pituitary fossa; on the floor of the skull; in the temporal, parietal, and occipital lobes; or in the midbrain, pons, or cerebellum all may produce signs detectable in the neuro-ophthalmologic examination. In addition, the examiner has available in the lids a means of estimating the condition of the body fluids and the thyroidal status, and he may observe septic emboli in the conjunctiva.

Ophthalmoscopy furnishes a sampling of the status of the arterioles of the rest of the body, particularly those of the brain and kidney. It permits estimation of the intraocular and intracranial pressure, and it provides a window for

the diagnosis of various other systemic diseases. Finally, ophthalmodynamometry even measures directly the blood pressure in the ophthalmic artery and the first branch of the internal carotid artery and assists in the accurate diagnosis of intracranial occlusive vascular disease.

SECOND CRANIAL NERVE

Establishing the integrity of the retina, optic nerves, optic chiasm, optic tracts, optic radiations, and occipital cortex is the most difficult and time-consuming part of the neuro-ophthalmologic examination. Here it is important to inquire into the history of visual difficulty, for it may be advisable to obtain detailed perimetric and tangent-screen examinations by an ophthalmologist skilled in these processes, even though no gross visual defects are found at the initial survey. Most lesions affecting the visual pathways can be detected by these procedures:

Measurement of visual acuity. A cardinal rule, which must never be disregarded, is that every defect in vision demands an explanation. The Snellen chart is the best method for measuring the integrity of the maculas and of the most numerous and most important fibers in the visual pathways. In testing of acuity, the glasses should be worn. The examiner should note the manner in which the patient reads the test letters, as small central scotomas and hemianopic lesions may be detected at this time. The small letters on the reading card should be seen easily. If the patient reads 20/20 on the Snellen chart and only 14/24 on the A.M.A. reading card or reads well on the near test and poorly on the Snellen chart, the visual difficulty is most probably of refractive origin. If deficiency exists in both tests, the cause may be an uncorrected refractive error; opacities in the cornea, lens, or vitreous; or disease in the retina or visual pathways.

Routine tests of color vision and of dark-adaptation are not necessary.

Examination of pupils. The pupils should be equal in size. Both the light reflex and the accommodative-convergence reflex should be test-

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ed. The latter is evaluated quickly by having the patient look first at the opposite wall and then at the examiner's finger. The responses of the two eyes should be approximately equal. If the vision is diminished by a lesion in the visual pathways, the light responses but not the accommodative-convergence reflexes will be depressed. If a patient is blind in both eyes from an infrageniculate lesion, the pupillary light responses will be absent; if the lesion is supragenigulate or the blindness is of psychogenic origin, the pupils will react to light. Lesions of the third cranial nerve, the ciliary ganglion in the orbit, or the midbrain cause derangement of the accommodative-convergence reflexes. A frequently disturbing cause of pupillary inequality is the slightly dilated, slowly reacting (myotonic, or Adie's) pupil, found in young women.

Ophthalmoscopy. Examination by ophthalmoscopy makes possible the detection of lesions of the lens and vitreous and furnishes a useful estimate of the degree of refractive error. Abnormalities of the retina often are associated with hereditary diseases of the central nervous system. The optic disks may be edematous or pale. Papilledema associated with good vision usually indicates choking of the disks, due to increased intracranial pressure; papilledema associated with poor vision is characteristic of optic neuritis.

Determination of visual fields. Most defects of the visual fields, except central scotomas or small peripheral defects, are demonstrable by the method of confrontation. This test must be done carefully and deliberately. The patient fixates his vision on the examiner's forehead, and a pen, a pencil, or a 3- to 5-mm. white bead on the end of a wand is brought from outside the patient's visual field toward the center until he first sees it. This action is repeated in the superotemporal, inferotemporal, superonasal, and inferonasal quadrants of each eye. Central scotomas may be suspected if there are defects in visual acuity not corrected by refraction. Any such defects should be plotted on the perimeter or tangent screen by an ophthalmologist.

THIRD, FOURTH, AND SIXTH CRANIAL NERVES

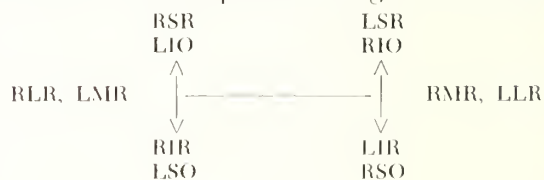
Recent lesions of these nerves almost invariably cause disability because of diplopia. The patient usually closes the eye on the affected side. Paralysis of conjugate lateral or vertical gaze rarely produces diplopia. Identification of the affected nerve is possible simply by inspection if the paralysis is complete. Partial paralysis of a nerve may be difficult to prove unless a red glass or a Maddox rod is placed before one eye while a small light is observed with both eyes open.

Complete paralysis of the third cranial nerve causes both vertical and horizontal separation of the two images. The upper lid of the affected eye is completely ptosed; its pupil is dilated and nonreactive; its accommodative power is lost; and, because of loss of function of the medial, superior, and inferior rectus and inferior oblique muscles, it deviates downward and laterally.

Complete paralysis of the fourth nerve produces its greatest disability when the eyes are turned downward for reading, as then the affected eye sees its image far below that of the normal eye. If the head is tipped forward and a little to the right or left, the diplopia may lessen or disappear.

Complete paralysis of the sixth cranial nerve causes horizontal separation of the 2 images, with least separation when the eyes are turned from the affected side. The image seen by the affected eye is displaced farther when the eyes are turned into the field of the paralyzed muscle.

Incomplete paralysis of one of these nerves is more difficult to diagnose. To examine the ocular rotations most efficiently, the eyes should be turned first far to the right and then up and down from this position; then the eyes should be rotated far to the left and again moved up and down. In this way, the eyes are placed in the optimal position for measuring the major action of each muscle. The diagram shows the pair of muscles tested in each of the 6 cardinal positions, with the patient facing the examiner.



In testing for small paralytic lesions with red glass or the Maddox rod, two points should be remembered: (1) the greatest separation of images will result when the eyes are turned into the field of action of the paralyzed muscle, and (2) the image displaced farther in the direction the eyes are moved belongs to the eye with the paralyzed muscle.

Transient conjugate deviation of the eyes toward, though sometimes away from, the hemiplegic side is common in cases of stroke affecting the cerebral hemispheres. A lesion in the pons is more likely to cause paresis of conjugate gaze toward the side of the lesion. An irritative lesion in a cerebral hemisphere causes deviation of the eyes away from the side of the lesion, while an irritative lesion in the pons causes deviation toward its side—in both instances for

only a day or two. A paralytic or atrophic lesion in a cerebral hemisphere causes paresis of conjugate gaze to the side opposite the lesion, while such a lesion in the pons causes paresis of conjugate gaze to the same side as the lesion, and such paresis may be of long duration.

Paresis of vertical gaze may result from a lesion affecting the quadrigeminal plate accompanied by a disturbance of the pupillary reflexes, convergence paralysis, or retraction nystagmus. Mild paresis of upward gaze without pathologic significance is seen often in the aged.

Nystagmus consisting of fast and slow components always results from a disturbance in the pons, cerebellum, labyrinth, or vestibular nerves. Care must be exercised not to misinterpret the small, ill-sustained nystagmoid movements, sometimes seen as a physiologic variant, or the rapid nystagmus, with small excursions, caused by the barbiturates. Nystagmus of labyrinthine origin is transient, is usually accompanied by vertigo, and beats in the same direction no matter which way the eyes are turned.

Nystagmus of cerebellar origin has a horizontal direction, its rest point is straight ahead, and the quick component is to the right on gaze to the right and to the left on gaze to the left. Nystagmus of pontine origin usually results from involvement of the vestibular nuclei or their connections. There are four main types: one is similar to that described as of cerebellar origin; another has its rest point 10 to 30° to one side, with rapid, small excursions beyond the rest point but slow, coarse movement on rotation to the opposite side; the third has a clockwise or counterclockwise rotary component, with the direction of the rotation maintained no matter which way the eyes are turned; and the fourth is vertical nystagmus, usually beating upward.

Lesions due to multiple sclerosis or other vascular origins often affect one or both median longitudinal fasciculi between midbrain and pons. They produce a peculiar dissociated nystagmus in which the outturning eye beats but the medial gaze or the inturning eye is paralyzed. Skew deviation of the eyes, in which they become vertically displaced, leading to vertical separation of the images, is due to a lesion in the pontine structures.

FIFTH AND SEVENTH CRANIAL NERVES

With a tiny strand of cotton fluff, the cornea is stroked lightly. This normally causes a violent blink reflex, and diminished sensation is easily detected. The fibers of the ophthalmic division of the fifth nerve supply the eye, upper lid, forehead, and top and side of the nose. The innerva-

tion of the lower lid is from the maxillary division of the fifth nerve. The cornea is in some danger when its sensation is lost, although the patient rarely has trouble with corneal ulceration unless the seventh nerve also is deficient.

The fibers of the seventh nerve to the orbicularis oculi emerge through the upper portion of the parotid gland. Paralysis of the nuclear or peripheral portions of the seventh nerve produces a slight ptosis of the upper lid and often ectropion of the lower lid; worst of all, the patient cannot close the eyelids. A supranuclear lesion rarely causes more than minimal paresis of the orbicularis oculi, even though the orbicularis oris may be completely paralyzed.

SYMPATHETIC AND PARASYMPATHETIC SYSTEMS

The dilator fibers of the pupil and Müller's muscles, the thin slips of smooth muscle in the upper and lower lids which maintain the tone of the eyelids, are supplied by sympathetic nerves which travel along the carotid artery and ophthalmic artery from the superior cervical ganglion. A lesion anywhere in the descending pathway in the brain stem and upper cervical cord or in the extramedullary fibers causes the pupil to be small, though reactive, and narrows the palpebral fissure. The latter effect is due to a loss of muscle tone, leading to elevation of the lower lid and slight ptosis of the upper lid.

The constrictor fibers of the pupil come from parasympathetic fibers in the lower division of the third cranial nerve. The afferent and efferent central and peripheral connections utilized in the light reflex are different from those of accommodation, even though each leads to constriction of the pupil. Regulation of lacrimation depends on connections with the parasympathetic fibers traveling through the seventh nerve and with sympathetic fibers from the carotid-cavernous plexus.

A FINAL WORD

It is obviously impossible in an article of this type to review the ophthalmologic findings in all disorders of the nervous system. Therefore, I would like to recommend Dr. Frank Walsh's book, *Clinical Neuro-ophthalmology* (Williams & Wilkins Company). This book will never be greatly out of date, and its reference material makes it valuable to all physicians.

In summary, I have attempted to present a plan of examination easily incorporated into every physical examination with the hope that it may be of value in the early discovery of intracranial disease.

Intravenous Angiography

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IN THE ROENTGEN and related literature since 1927, when Egas Moniz¹ described the practical and significant in vivo visualization of the vascular bed of the cranial vault, there are reams of articles about the introduction of various contrast media via innumerable routes, both venous and arterial, enabling visualization of entire vascular circulatory routes or selective isolated viscera. It is not the purpose of this report to review the many methods nor to classify them according to their merits. Each may have a unique and preferential position, depending on the specific need at the time. The purpose of this discussion is to reemphasize the value of the once-discarded method of intravenous angiography based on the approach of Bernstein and associates² and Greenspan and associates.³ This procedure has a definite place in the preoperative diagnosis of vascular lesions not only in large university hospitals but also in smaller general hospitals where complete patient care and treatment are available.

Intravenous aortography was not a new idea even when Weens and associates⁴ suggested it for nephrographic studies of renal lesions in order to avoid the hazards of translumbar aortography.⁵⁻¹² The latter method was more or less abandoned because of direct damage to kidney or cord by high, momentary contrast concentrations.

It is not difficult to see why these earlier suggestions did not gain immediate popularity, for the contrast used was usually 70 per cent Diodrast, in doses of 30 to 50 cc., introduced by hand-injection methods. Thus, vascular bed visualization was understandably faint in many instances.

With the introduction of higher concentrations of iodinated contrast media,¹³ interest in the method was understandably revived. Bernstein and associates² and Greenspan and associates,³

at the University of Minnesota Hospitals, were keen to appreciate its value in the preoperative localization of aneurysms, blocks, and so forth. They felt that intravenous aortography would decrease the instrumentation necessary for routine selective surgical or percutaneous angiography in the older age group, in which surgical repair of vascular defects is most frequent. Likewise, in iliac blocks, catheterization of diseased vessels is often contraindicated. Thus, by rapidly injecting large quantities of highly concentrated contrast media using a rapid film changer, the practicality of intravenous angiography was demonstrated.

Our experience has convinced us that this method has furthered vascular surgery in a well-equipped, small general hospital as well as in a large university center. We feel that selective angiography is the method of choice in cardiology, aortic valvular disease, and peripheral vascular visualization. The intravenous method is considered an excellent, fast diagnostic procedure in delineation of the aorta and its main tributaries.

TECHNIC

Selection of contrast medium is obviously one of the most important phases, for before the high-concentration iodinated compounds were available, this procedure was not an ideal means for studying adult subjects. In order to deliver a

DELAY BETWEEN INJECTION OF CONTRAST MEDIUM AND EXPOSURE

Location	Amount of delay (seconds)
Right heart and pulmonary artery	None
Pulmonary veins, left atrium, and left ventricle	2 to 3
Ascending aorta and arch	5 to 6
Thoracic aorta and upper abdominal aorta	9 to 10
Aortic bifurcation	10 to 11

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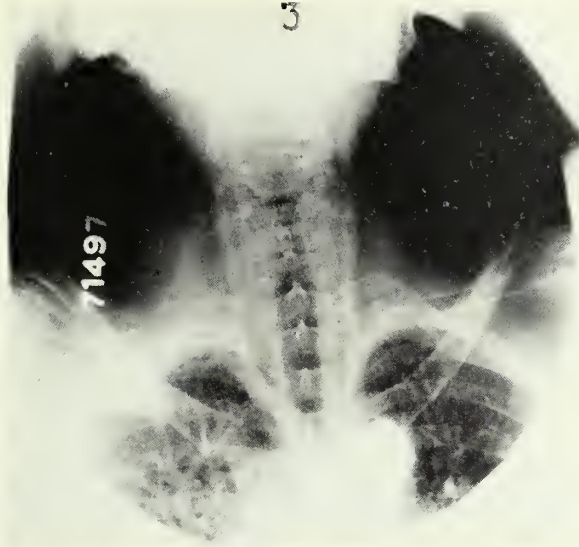


Fig. 1. Clinical question of common carotid syndrome excluded by examination of common carotids. Vertebral vessels well visualized.

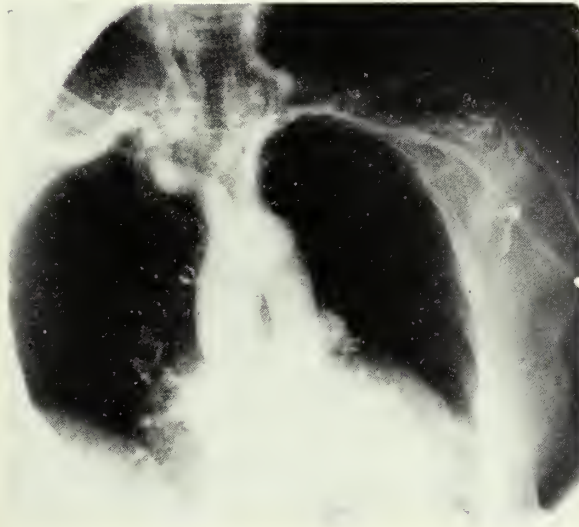


Fig. 2. Investigation of cold, painful left upper extremity with almost complete absence of left pulse. (left) Partial attenuation of 6 to 7 cm. of left subclavian artery by multiple plaques with associated demonstration of collaterals and decreased caliber of peripheral subclavian artery as compared with normal right side. (above) An incidental finding was that of kinking of the innominate artery which, on the plain film, simulated a mass.

sufficiently concentrated bolus to the left heart and aorta, either 90 per cent Hypaque or 85 per cent Renografin is used. The volume used ranges from approximately 1 to 100 cc. per kilogram. The average dose for an adult varies between 70 and 80 cc.

As might be assumed, the speed of injection is almost as important as the choice of the contrast medium. It is injected via a catheter inserted through the antecubital vein for a distance of 4 to 8 in. The catheter should be of the largest caliber that can be inserted into the vein. The entire bolus is injected in one to two seconds, depending upon the caliber of the catheter, using an Elema-Schönander mechanical injector.

We use the Decholin circulation time rather

than the radioactive Renografin circulation time.^{2,3} This time is used as a guide to the delay between the injection of contrast medium and the beginning of the exposure sequence. The times in the table have proved to be usual in an average-sized patient. The time between the injection and the beginning of the exposures permits the operator to leave the room. A Schönander rapid film changer is used at a rate of 1 to 1.5 exposures per second.

The patient is adequately sedated before being taken to the x-ray department. A scout film is taken and, during its processing, the circulation time is determined and the cutdown performed. During these procedures, the patient is told about the flush reaction that he or she will ex-

Fig. 3. (*below*) Routine chest film shows large left paramediastinal mass which, on fluoroscopy and kymography, proved to be vascular. Based on roentgen and clinical findings, this was interpreted as a coarctation change. (*right*) Aortogram proved the coarctation, but the vascular mass was actually the large, dilated, and tortuous left subclavian artery.

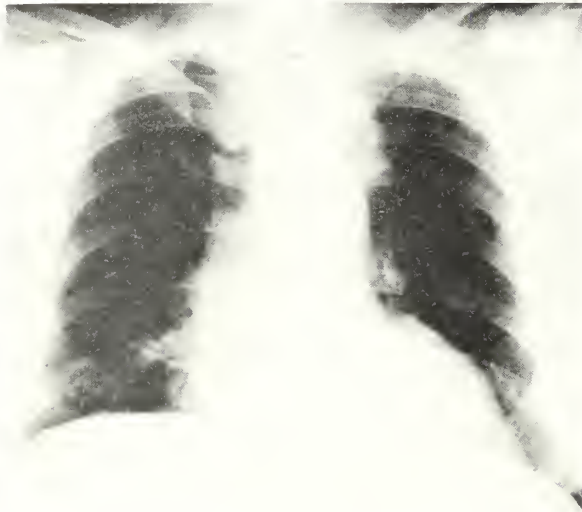


Fig. 4. (*left*) Chest film of a middle-aged patient who had had known cardiac infarctions during past year with recent appearance of localized left ventricular ectasia. (*above*) Contrast injected intravenously delineated the extent of ventricular aneurysm, which was successfully excised.

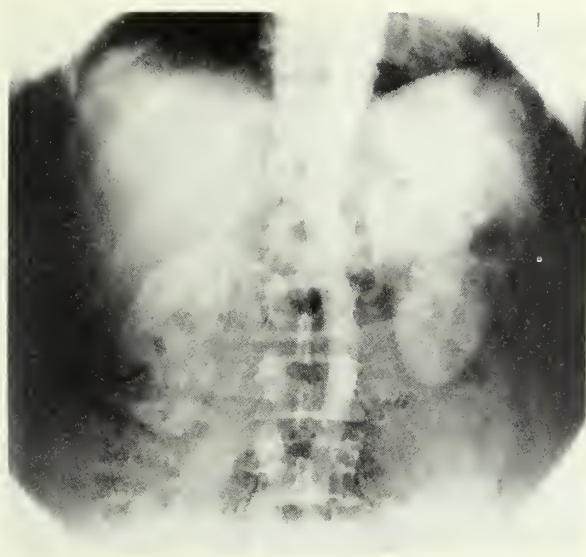


Fig. 5. Normal intravenous abdominal aortogram showing major and minor branches.

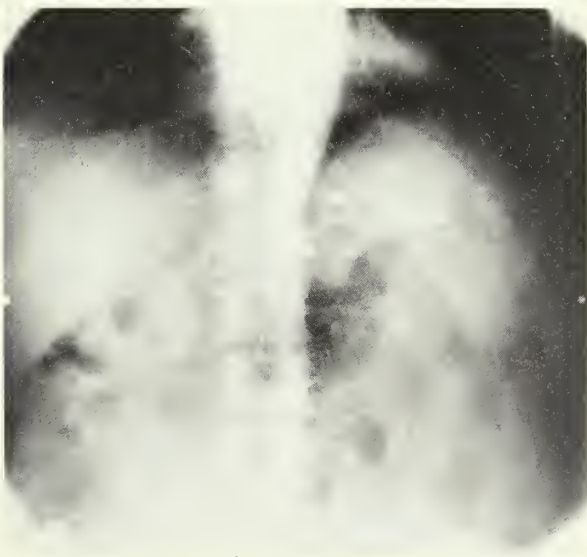


Fig. 6. (*above*) Pyelogram showing silent filling defect of upper pole of left kidney, which was found on routine physical work-up. No collecting system invasion was apparent. (*right*) Angiogram shows abundant vascular bed to mass during late stain phases, indicating diagnosis of neoplasm.



Fig. 7 (left) Large subcarinal mass superimposed on heart. (below) On intravenous angiogram, the mass showed its complete independence from vascular structures that compressed the atrium and elevated the right pulmonary artery. Preoperative diagnosis was subcarinal benign cyst and was confirmed at surgery.

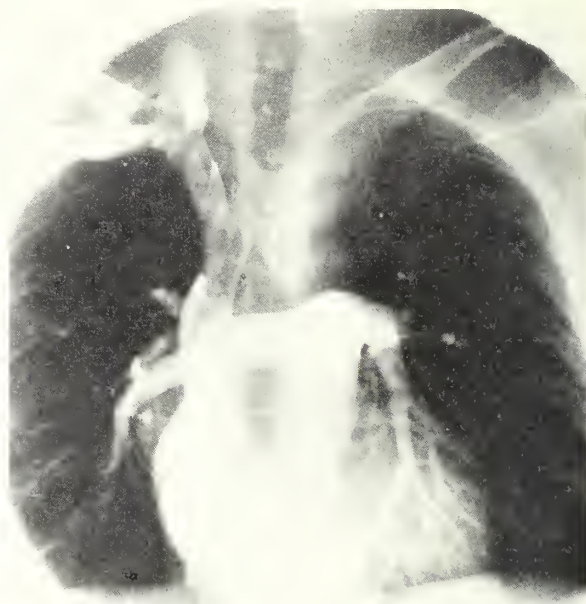


Fig. 8. Intravenous angiography in case of gradually increasing idiopathic hypertension showed small left renal artery as compared with right. In addition, left nephrogram showed definite difference in density when compared with normal right side.



Fig. 9. Clinically palpable abdominal aortic aneurysm was shown to extend from below renal arteries down to and including part of right common iliac artery and 6 to 8 cm. of left common iliac artery. Aneurysm was removed and replaced with a prosthesis.

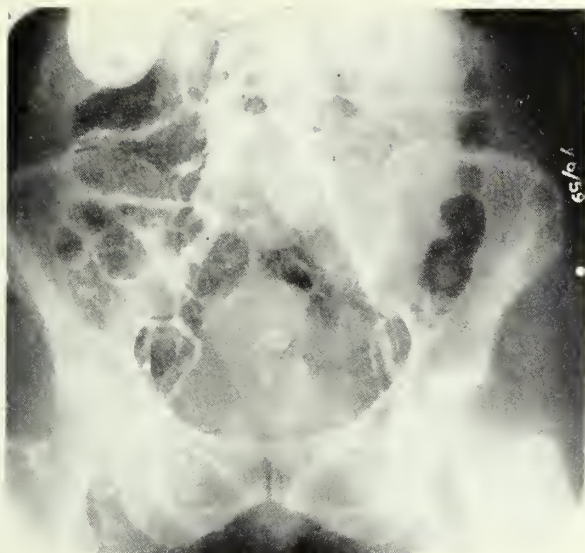


Fig. 10. Clinical absence of right femoral pulsations prompted angiographic examination, which showed partial block of right distal aorta and external iliac artery. Left common iliac and its branches showed multiple plaques encroaching upon lumina. Right femoral artery was filled by multiple collaterals.

perience. The examination is then completed quickly after the preliminaries are finished.

As previously intimated, use of a rapid film changer and contrast injector is considered the method of choice. If these are not available, the procedure can be performed, although with obvious technical drawbacks, using Steinberg and associates'¹⁴ method of simultaneous injection through both antecubital veins and using handchanged cassetts. This is obviously subject to greater chance insofar as speed of introduction of the contrast and the exact timing of exposures are concerned.

We have adopted the procedure of intravenous angiography for the diagnosis of defects of the aorta and its tributaries as well as abnormalities of its end organs. To date we have studied more than 50 cases which fall into this category.

In cases of the common carotid syndrome, this method offers a simple approach to the delineation of the carotids and vertebral arteries (figure 1). Carotid kinks, great vessel aneurysms of the paramediastinal level, and solid masses at this level can be differentiated (figure 2 *a* and *b*). In addition, it is an excellent method for evaluating the type and extent of subclavian defects. We do not feel that it is a suitable approach for the study of cerebral circulation. It has proved very helpful in identifying coarctations as to type, location, and associated vascular changes

(figure 3 *a* and *b*). Even though we have not used this method as the one of choice in cardiac studies, we did find that, in a case of cardiac aneurysm in which catheterization was considered hazardous, it delineated the true size of the aneurysm, which was successfully excised and repaired (figure 4 *a* and *b*). Needless to say, it is our present method of choice in delineating thoracic aortic aneurysms, even though they are not included in the illustrations.

As might be anticipated, this is an ideal survey method for delineation of the abdominal aorta and its tributaries (figure 5). We advocate its use in differentiation of cyst and neoplasm diagnoses in renal problems (figure 6 *a*, *b*, *c*, and *d*). While referring to solid, nonvascular masses, one might regress to the mediastinum, where the examination can be used to differentiate between a solid and apparently cystic mass (figure 7 *a* and *b*). In cases of idiopathic hypertension, intravenous angiography may delineate renal vessel abnormalities or decreased renal blood flow (figure 8). As in other methods of aortography, it is used to delineate the entire zone of involvement of an abdominal aortic aneurysm and its encroachment on other vessels (figure 9). It goes without saying that it is definitely the simplest way to evaluate iliac or femoral blocks in the older age group, where instrumentation is contraindicated (figure 10). We have also used it to determine gross peripheral femoral run-offs. If suggestive for defect, we follow up with direct femoral arteriograms.

As an academic aside, we were impressed with the relative staining of the osseous bed in an isolated case of Paget's disease, which we were investigating for femoral blocks.

CONCLUSIONS

The employment of intravenous angiography for diagnosis and evaluation of the nature of surgical vascular lesions and adjacent visceral lesions is advocated, particularly in cases in which instrumentation should be kept at a minimum. We advocate it not only for large vascular centers but also for well-equipped smaller general hospitals. Our choice is that of the rapid injection of large quantities of highly concentrated contrast media coupled with rapid film changer technics.

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ALTHOUGH BLOOD ELEMENTS are not altered significantly during and after minor operations, extensive surgical procedures cause considerable changes.

During major operations, total leukocytes increase rapidly to about 105 per cent above preoperative levels; eosinophils decrease to about 88 per cent and platelets to about 46 per cent below preoperative values. All these blood elements gradually return to normal by the fifth postoperative day. Then, while leukocyte levels remain unchanged, platelets increase to about 29 per cent and eosinophils to about 88 per cent above preoperative levels by the ninth day, returning to normal by the fourteenth day.

The similar and simultaneous behavior of eosinophils and platelets probably is caused by the varying reactions of the pituitary-adrenal axis to surgical stress.

Sudden and pronounced thrombocytosis usually appears on the third to tenth days, the period when postoperative venous thrombosis usually is observed. Cause of the thrombosis may be the sudden rise of platelets, in association with pre-existing endothelial damage and diminished circulation.

H. PEPPER and S. LINDSAY: Responses of platelets, eosinophils, and total leucocytes during and following surgical procedures. *Surg., Gynec. & Obst.* 110:319-326, 1960.

SUDDEN, PROFOUND ionic imbalances may cause ventricular fibrillation during operation, while cardiac arrest results primarily from anoxia. The most ominous sign of arrest is bradycardia.

As a result of such stimuli as anoxia, stress, reflex reactions, hemorrhage, and excitation, the sympathetic nervous system or adrenal glands act through nervous pathways to liberate the potassium-hexose-phosphate complex from the liver. If these stimuli act rapidly, potassium suddenly shifts into the blood stream and goes directly to the heart. The abrupt appearance of excessive amounts of potassium in the extracellular compartment results in large differences in electric potential across the myocardial cell membrane.

With low concentrations of potassium, only the rate and rhythm of the heart are affected. When 20 mg. of potassium per kilogram of body weight is injected intravenously into dogs in two to four seconds, local areas are triggered by differences in potential, resulting in ventricular fibrillation. With extremely high concentrations, the myocardium apparently is depolarized suddenly and is overwhelmed into arrest by the massive transfer of ions.

Serum potassium rapidly returns to normal, because the ion is quickly transferred back to the liver.

The various factors inducing potassium release can act simultaneously or sequentially to produce summation. Some of these factors, along with drugs and anesthetic agents, sensitize, stimulate, irritate, or depress the myocardium, thus promoting further synergism.

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Peptic Ulcers in Children

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PEPTIC ULCER IN CHILDHOOD is no longer a diagnostic curiosity. In the last ten years, 95 children with ulcer were diagnosed radiologically by our group.¹ The apparent recent increase in incidence of childhood ulcer is undoubtedly due to 2 factors: an increased index of suspicion on the part of the clinician and radiologist and improved spot film equipment. Goldsberry's prediction² of 50,000 undiagnosed gastric ulcers in the 1 to 6 age group suggests that this diagnosis should be suspected.

The incidence of peptic ulcer in children varies from a low of 0.3 of 1 per cent³ to 1.5 per cent⁴ in surgery and autopsy series to a high of 12 per cent⁵ in radiology series. The true incidence must be somewhere in between, because patients coming to autopsy or surgery and patients whose symptoms require roentgenographic examinations do not represent a normal, healthy segment of the population. In our series, about 10 per cent of children's stomach examinations each year have revealed an ulcer. The number of children examined per year has increased as we have become more aware of the problem. In 1949, only 2 ulcers were diagnosed in 20 pediatric stomach examinations; in 1959, 16 new ulcers were found in 160 stomach examinations.

More ulcers have been noted in the first year of life than in any other year because of their spectacular manifestations—bleeding and perforation. We have had 12 ulcers appearing in infants under 1 year of age. Hematemesis due to a duodenal ulcer developed in one child twelve hours after birth. Our age distribution is in agreement with Morgan's⁶ statement that the incidence per year is probably about equal but that cases in other years do not often come to surgery or autopsy.

In compiling the figures for this series, each case has been counted only once. Some patients have had repeated duodenal ulcers or both duodenal and gastric ulcers at different times.

Table 1 shows our figures for duodenal ulcer. There were 52 males and 36 females with duodenal ulcer. This male predominance agrees with the other series reported. There were 7 patients with gastric ulcer (table 2). This ratio of 7 gastric ulcers to 88 duodenal ulcers is about the same as that for adults reported in the literature. In contrast to the low incidence of patients with a positive family history of ulcer noted by Muggia and Spiro,⁷ our percentage was 40 (table 3), which is probably due to the well-known familial occurrence of ulcer. One 5-year-old had 3 siblings, both parents, and an uncle with ulcers.

We did not make the diagnosis of peptic ulcer unless the crater was definitely visualized. Some authors have included cases in which indirect signs of ulcer have been present or clinical symptoms have been very suggestive. We prefer to report cases with hypersecretion, pylorospasm, and irritability of the duodenal bulb as showing these findings rather than as peptic ulcer. Cases fulfilling Kirklin's⁸ definition of duodenitis—exaggerated mucosal folds in the bulb, with spastic deformity, irritability, and localized tenderness—are reported as duodenitis. Three of the cases originally diagnosed as duodenitis, were found upon reexamination to have a duodenal ulcer after the irritability and deformity had responded to ulcer treatment. In the literature, the incidence of duodenitis is about one third that of duodenal ulcer; in this series, it is 38 to 88, or 43 per cent. Male predominance is also present in duodenitis (table 4).

Morgan's article⁶ covers the technic of examination very thoroughly. A short visit with the patient, before the lights are out, to gain his confidence will shorten the total examination time. Patient exposure is minimized by using not more than two to three minutes of fluoroscopy and by relying on 8 to 12 spot films for the diagnosis. The craters are often too small for fluoroscopic visualization. Some patients will require sedation or mummification before they can be examined. Water-soluble contrast materials are more suitable than barium for very young patients.

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TABLE 1

AGE AND SEX DISTRIBUTION OF DUODENAL ULCER

Age	Male	Female	Total
0 to 14 days	0	1	1
14 days to 1 year	6	3	9
1 to 6 years	8	9	17
6 to 11 years	18	15	33
11 to 16 years	20	8	28
	52	36	88

TABLE 3

FAMILY HISTORY OF ULCER

	Number	Percent
Duodenal ulcer	37 of 88 positive	40
Gastric ulcer	1 of 7 positive	
Duodenitis	5 of 38 positive	13

TABLE 4

AGE AND SEX DISTRIBUTION OF DUODENITIS

Age	Male	Female	Total
1 to 6 years	3	3	6
6 to 11 years	11	7	18
11 to 16 years	9	5	14
	23	15	38

TABLE 2

AGE AND SEX DISTRIBUTION OF GASTRIC ULCER

Age	Male	Female	Total
0 to 14 days	1	0	1
14 days to 1 year	1	0	1
1 to 6 years	0	1	1
6 to 11 years	1	2	3
11 to 16 years	1	0	1
	4	3	7

The symptomatology of pediatric ulcer is quite variable. In the first year, the symptoms are usually dramatic—hemorrhage, shock, or profound anemia. The preschool child usually presents a feeding problem. Symptoms in the school-age child may be vague, or definite ulcer symptoms, as are seen in the adult, may be present.

CASE REPORTS

The case histories presented here have been selected to bring out other facets of peptic ulcer.

Case 1. A pale, 6-month-old male was referred with a history of spitting up feedings. Examination revealed the stools to be positive for occult blood. There was no family history of ulcer. Roentgenograms revealed a gastric ulcer (figure 1). The symptoms cleared rapidly upon treatment with transfusions and antacids. Three months later, the infant was symptom-free and, on reexamination, the ulcer was healed.

Case 2. This 3-year-old male was admitted because of

Fig. 1. *Case 1.* Antral ulcer in 6-month-old male.Fig. 2. *Case 2.* Duodenal ulcer in 3-year-old male.

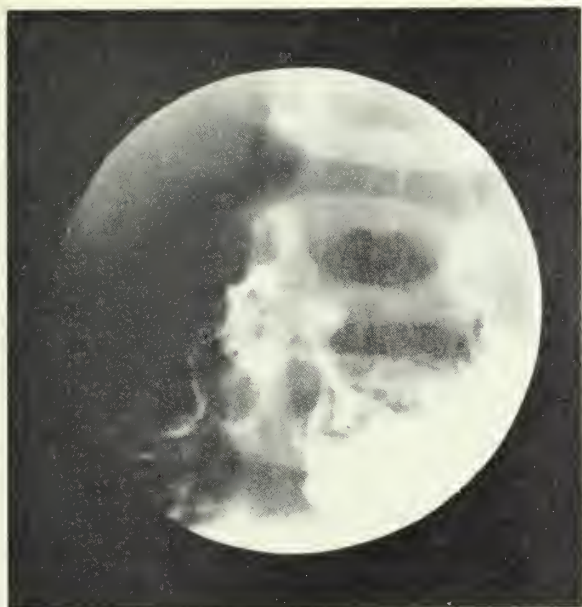


Fig. 3. *Case 3.* (left) Duodenal ulcer in 4-year-old female.

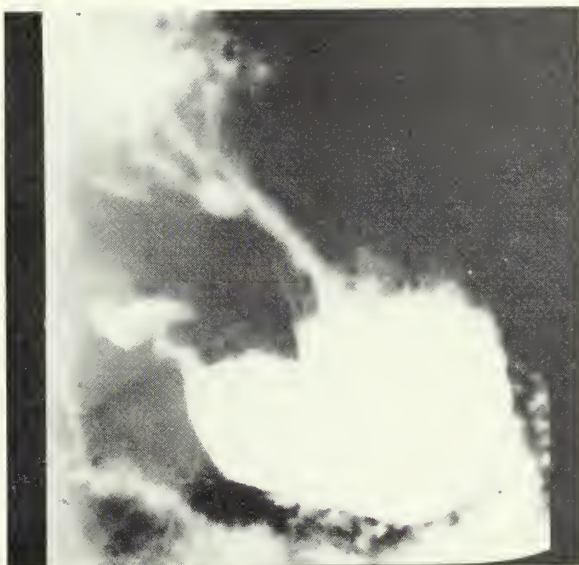
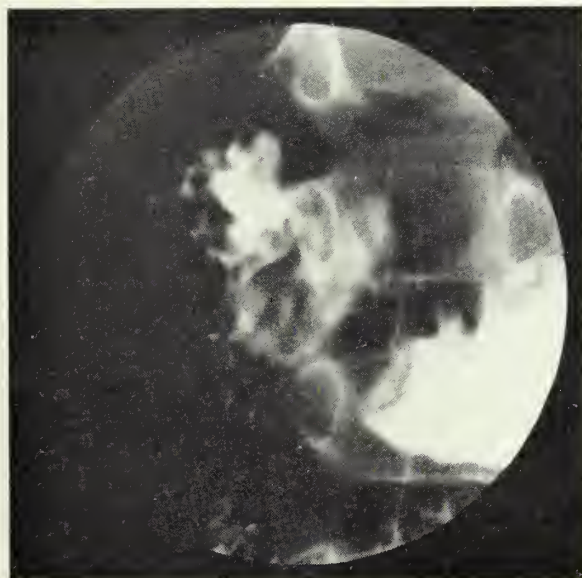


Fig. 4. *Case 4.* (above) Lesser curvature gastric ulcer in 5-year-old female.

Fig. 5. *Case 5.* (left) Duodenal ulcer in 6½-year-old male.

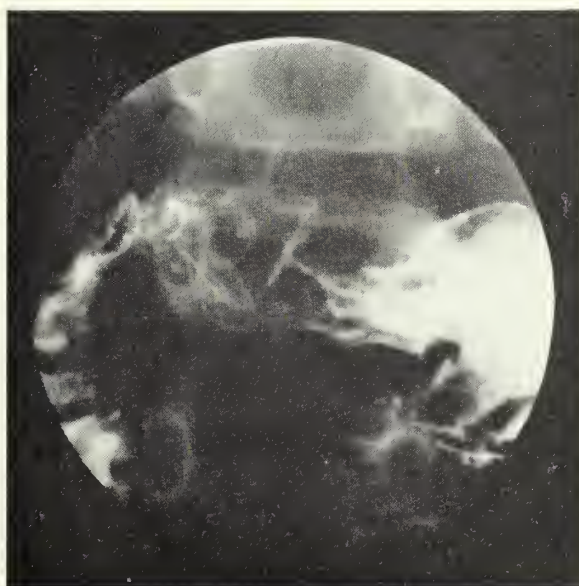


Fig. 6. *Case 6.* Duodenal ulcer in 7-year-old male.

fever and vomiting of twenty-four hours' duration. He had had epigastric pain, usually at mealtime, for one year. His father was being treated for a duodenal ulcer. A large ulcer crater was seen in the duodenal bulb (figure 2). The child's symptoms cleared with antacids, and he has not had a stomach pain since.

Case 3. A 4-year-old female was examined because of pallor, dark circles under the eyes, and stomach-aches of six months' duration. There was no family history of ulcer. Hemoglobin was 75 per cent. Roentgenograms revealed a duodenal ulcer (figure 3). The patient responded well to treatment and has been symptom-free on subsequent visits.

Case 4. Cyclic vomiting and poor appetite were the presenting symptoms of this nervous 5-year-old female. Initial x-ray films revealed pylorospasm and hypersecretion. There was no family history of ulcer. Her symp-



Fig. 7. *Case 7.* Duodenal ulcer in 8-year-old female whose original examination revealed duodenitis.

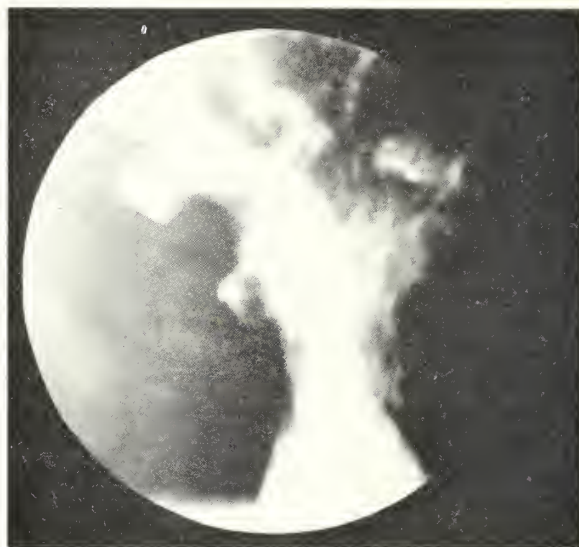


Fig. 8. *Case 8.* Gastric ulcer in 10-year-old male, which developed three years after treatment for duodenal ulcer.

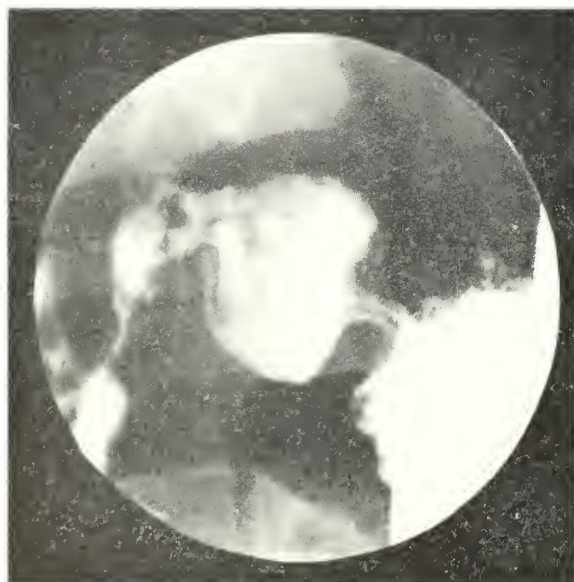


Fig. 9. *Case 9.* (left) Duodenal ulcer in 10-year-old male.

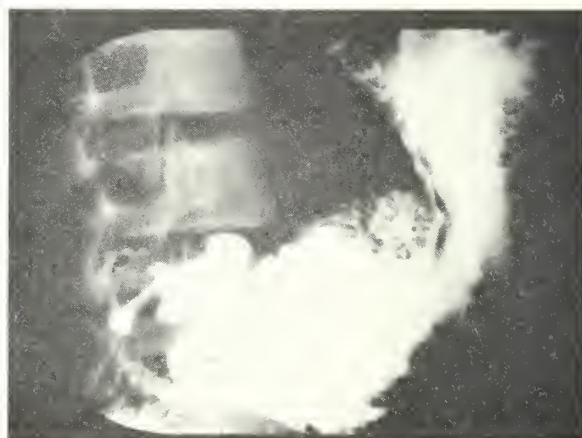


Fig. 10. *Case 10.* (above) Two duodenal ulcers in 14-year-old male.

tous continued despite antacid therapy. One year later, repeat examination revealed a gastric ulcer on the lesser curvature (figure 4). Atropine, Amphojel, and a bland diet relieved the symptoms. Follow-up examinations during the next four years were normal. The child still vomits when she is disappointed, however.

Case 5. This nervous, well-developed 6½-year-old male was examined because of nausea and abdominal discomfort. Two years previously, he had been seen in another city with similar symptoms. Examination at that time revealed a duodenal ulcer, which responded satisfactorily to treatment and was improved on later examination. His father has had peptic ulcer. The present x-ray film also reveals a duodenal ulcer (figure 5). He has not yet returned for reexamination.

Case 6. A 7-year-old male was referred with a history of stomach-aches occurring one hour after meals, most marked in the mornings and localized to the periumbilical region. His older brother has a peptic ulcer. Spot films show a crater in the middle of the duodenal bulb (figure 6). After ulcer treatment, weight gain and symptomatic improvement were noted on subsequent visits.

Case 7. A history of stomach-ache and headache of one month's duration led to the admission of this 8-year-old female. There had been no emesis or diarrhea. Family history was negative for ulcer. Roentgenograms revealed hypertrophy and irregularity of the duodenal mucosa but no definite ulcer crater. Pinworms were found in the anus. The patient was discharged on an ulcer regimen. Two months later, there was marked symptomatic im-

provement, but a definite ulcer crater was found in the distal bulb (figure 7). Her ulcer management was then intensified, and she has been symptom-free since.

Case 8. A 7-year-old male was examined because of preprandial distress and early morning epigastric pain, present for two days. The distress was relieved by food. His father had a duodenal ulcer. Hemoglobin was 66 per cent. Roentgenologic examination revealed a duodenal ulcer. The boy's symptoms cleared and his hemoglobin returned to normal under treatment. Three years later, he was seen because of fatigue, pallor, and stomach pain. An ulcer crater was present high on the lesser curvature of the stomach (figure 8). This healed slowly but completely with treatment. One year later, the symptoms recurred and a similar crater was found. He has been kept on a strict ulcer regimen for the past three years and has remained asymptomatic.

Case 9. This 10-year-old-male was admitted with a fever, sore throat, cough, diarrhea, and intermittent low abdominal pain. Symptoms had been present for one week. There had also been 1 emesis daily. Three uncles had duodenal ulcer. Chest films and plain films of the abdomen were normal. White blood count was 15,000. All symptoms, except abdominal pain and emesis, cleared after penicillin treatment, and white blood count returned to normal. Stomach examination revealed an ulcer crater in the duodenal bulb (figure 9). Emesis and abdominal pain stopped when ulcer therapy was started. Eight months later, the boy was again hospitalized for epigastric pain and emesis. He had not followed his diet for three months preceding the second admission. He is now symptom-free on an ulcer regimen.

Case 10. An apprehensive 14-year-old male was admitted because of upper abdominal pain and frequent emesis, present for two weeks. He had had many similar episodes during the preceding seven years. Because of similar attacks, a tonsillectomy had been performed at age 10 and an appendectomy at age 12. He stated that he always became sick in science class at school. His poorest grades were in science. His father had had a peptic ulcer for one year. His mother constantly complained of her stomach. The duodenal bulb revealed 2 ulcer craters (figure 10). Symptoms cleared rapidly

under therapy. The ulcers healed, and the duodenal bulb gradually returned to normal on later examinations.

DISCUSSION

The 95 patients observed by us suggest that the occurrence of peptic ulcer is more common in children than is usually realized, but this diagnosis should not be made unless the crater can be demonstrated. Food allergy, mesenteric adenitis, acute and chronic appendicitis, dietary indiscretion, abdominal epilepsy or migraine, gastroenteritis, acute pyelonephritis, volvulus, and intussusception can cause the same symptoms and must be considered in the differential diagnosis. We believe any child with repeated episodes of abdominal discomfort or symptoms of long duration would benefit from a careful radiologic examination of the stomach.

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COLECTOMY WITH ILEOSTOMY is the preferred treatment for nonspecific ulcerative colitis. Resection with ileoproctostomy should be reserved for the small group of patients in whom [1] colitis is confined proximal to the descending colon and [2] the rectum and sigmoid are and have been free of disease.

Of 468 patients with ulcerative colitis treated between September 1945 and December 1958, 124 had colectomy. Colitis had prevailed for five or more years in 50 of the operated patients; 23 had acute fulminating disease. Major associated conditions in the operated group included pseudopolyposis in 54.8 per cent, anorectal abscess or fistula in 43.4 per cent, ileitis in 41.4 per cent, and intestinal carcinoma in 11.3 per cent.

Death during or immediately after colectomy occurred in 4 per cent. Another 4.8 per cent died within a year after discharge from the hospital. Of the 23 patients with acute fulminating disease, 18 were alive and well three months to ten years after colectomy.

Exclusion of the 14 patients with cancer and 31 patients who had had operation less than five years previously left a total of 79 for calculation of survival and rehabilitation. Of these, 58 survived five and 21 survived ten years or more.

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Viruses and the Public's Health

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VIRUSES are the most notorious cause of communicable disease today. They account for the lion's share of current infectious illness, ranging from the commonplace and mild to the rare, crippling, and deadly. The viral diseases of childhood—measles, mumps, chickenpox, and rubella—are accepted as a part of growing up and are considered alarming chiefly when they occur outside the usual age limits or when they pave the way for serious complications and chronic conditions. Others, particularly those that attack the central nervous system, such as rabies, encephalitis, and poliomyelitis, spring to mind automatically whenever the term dread diseases is mentioned.

Few, if any, parts of the body seem to be safe from these highly selective, intracellular parasites. Some attack the brain and central nervous system, others the respiratory tract, still others the digestive tract, and so on down the line. There are even species that produce plantar warts. All living things—man, animals, and plants—are confronted with an array of viruses that have a unique preference for them as hosts and for specific systems within them. Even bacteria can be destroyed by tadpole-shaped viruses, called bacteriophages.

Viruses, by their very nature, have presented obstacles to study. The majority of them have been unknown, unseen adversaries, manifesting themselves chiefly by what they do and how they spread. Consequently, they have been poorly understood. The contrast between our basic knowledge of viruses and our knowledge of bacteria shows up conspicuously in the degree of control of each that has been achieved. Through accurate diagnosis, general and specific preventive activities, and chemotherapy, the sting has been removed from many bacterial diseases. Of course, there is still uncharted territory to be

explored, interpreted, and conquered in this category, also. Those persons who work in the tuberculosis field or with other bacterial diseases are aware of the unfinished business that stands between them and the goals they are seeking to achieve. Nevertheless, taken as a whole, bacteria represent a diminishing threat to human life.

The situation is quite different with regard to the viral diseases. Spectacular triumphs against a few individual viral diseases stand out in strong relief to the general rule. For instance, we have enough information on some to control them through immunization of human and animal hosts or through control of insect vectors and environmental factors. Oddly enough, the first types of immunization ever to be developed were directed against viral diseases—innoculation and vaccination for smallpox and prophylactic treatment for rabies. For most viral diseases, however, the fund of knowledge is still inadequate to the task. At present, only a few are preventable and even fewer—principally those of the psittacosis-lymphogranuloma group—are amenable to chemotherapy.

Elaboration of control measures for any infectious disease usually awaits precise knowledge of the causative organism, its ecology, and its modes of spread. We are just beginning to strip viruses of the obscurity that has sheltered them in the past. Under the spotlight of increasing intelligence, earlier concepts of viral diseases are enlarging and sometimes changing and the number of known pathogenic viruses is skyrocketing. At the turn of the century, only about a dozen human viruses were known; fifteen years ago, there were probably not more than 70; today, there are more than 150, with new ones coming up regularly. At last, we are beginning to hurdle series of obstacles that stood in the way of understanding these elusive pathogens. In this fact lies the expectation that we will soon accumulate sufficient knowledge to tip the balance against an increasing number of viral diseases.

The word virus originated during the Middle Ages to denote a venomous exudate from

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wounds. Later, invisible substances were found to be the cause rather than the result of infective processes. With acceptance of the germ theory of disease, bacteria were isolated, cultured, examined under the microscope, identified, and associated with specific disease conditions. Their ecology was studied, their modes of spread were determined, and rational methods of control were developed. However, there remained a number of infectious diseases for which no bacteria could be found. Bacteriologists found that infective particles, small enough to pass through the pores of porcelain filters, could produce disease. The term filterable virus was applied to these pathogenic substances for many years. Because the tools of the bacteriologist, chemist, and physicist were inadequate to penetrate their secrets, viruses remained invisible and inscrutable. They were distinguishable by their capacity to spread and produce disease.

Viruses have many qualities that set them apart from bacteria. Most of them are too small to be examined under the ordinary microscope and identified by their morphologic characteristics. They cannot be grown in culture media suitable for bacteria, since they multiply and take on the attributes of life only within susceptible living cells. They cannot be tasted, smelled, touched, or measured by methods commonly used for other pathogens. Like ideas, they are intangible, pervasive, and able to spread and bear fruit only among those who are susceptible to them. To carry the comparison a step further, they are equally difficult to defeat. That is why we speak of the virus of ideologies, political and social, that are in opposition to our way of life.

The first real breakthrough in the scientific study of viruses was the discovery, years ago, that some human and animal disease processes could be reproduced in the suckling white mouse and that virus could be cultivated and harvested in the embryonic chicken egg. The mouse has been joined by more expensive and complicated animals, such as the Rhesus monkey. However, we are still handicapped by the lack of convenient intact laboratory animals to serve as substitute hosts for most of the human viral diseases.

Until recently, cultivation and isolation of viruses was a cumbersome, tedious, and expensive procedure. Advances in tissue culture methods have now placed it on a more practical footing. Use of tissue cultures for the propagation of viruses dates back to 1913. Inherent problems limited their use to the research laboratory. In 1949, Enders and his associates reported

growing poliovirus in monkey kidney cells, the first successful attempt to cultivate it in non-nervous system tissues. Since then, several kinds of tissue cells have proved suitable for the propagation of one virus or another. Monkey kidney, human amnion, HeLa, and other cells are now widely used for the growth and differentiation of viruses. The ordinary microscope and antibiotics have found their niche in virology. The destructive changes that occur in infected tissues can be seen and studied under the low-power microscope. Antibiotics, which are of no avail in the treatment of most viral diseases, serve as an adjunct to virus studies by keeping down bacterial contamination in tissue cultures.

Serologic work on viruses has also advanced, with laboratory techniques developing around specific antigen-antibody systems. Most of these tests are not yet specific enough for clinical or epidemiologic purposes. They provide evidence of past infection, which is useful in constructing a profile of previous viral experience within a community. So far, it has not been possible to distinguish between the antibodies acquired by natural infection, such as poliovirus, and those induced by vaccine.

Some interesting questions have been raised as a result of finding rabies antibodies in healthy, trapped wild animals. Bats have been known to carry the virus for months without developing clinical symptoms of the disease, but they were thought to be exceptional in this respect. How and when other animals received their exposure to the virus is unknown. Since we have no knowledge of subclinical rabies infections, the significance of the finding is not understood at this time.

A number of serologic tests for viruses are being studied in conjunction with the fluorescent antibody technique for rapid diagnosis. The diagnostic test for rabies has been field-tested and found specific and practical enough for wide spread use, with results obtainable within a day. To the bite victim and the physician or public health worker who must east the die for or against the Pasteur treatment, this time-saving element is of inestimable value.

The electron microscope, with a magnification power of up to 200,000, has brought viruses within the range of vision. A technique called metal shadow casting provides the necessary contrast between the background material and the viral particles. Many investigators have photographed different viruses of varying sizes and shapes. Each follows its own geometric pattern; the influenza virus, for example, looks like a fluffy

powder puff. The antibody molecule, which immunochemists believe is a few millimicrons smaller than the encephalitis virus, may someday be seen.

Scores of previously undiscovered viruses have been identified within the past few years. When so many new microorganisms come to light so rapidly, it takes time to sort the pathogens from the harmless saprophytes and to determine whether they are agents of known or new diseases. A virus is usually considered pathogenic when it is frequently found during a characteristic illness, when specific antibodies develop in the host, and when the isolated virus produces a similar disease picture in other susceptible hosts.

As new viruses have been identified and associated with disease states, many have been found to produce signs and symptoms that formerly pointed to other agents. This characteristic is shared with other kinds of pathogens, also. In suspected tuberculosis, the culprit may turn out to be a fungus, one of many atypical acid-fast bacilli, or something else quite distinct from tubercle bacilli. Wisdom dictates that a clinical diagnosis of poliomyelitis be followed by a question mark until the presence of poliovirus is confirmed in the laboratory, because the symptoms may be caused by one of the other enteroviruses. The encephalitis syndrome requires careful definition of its source. It may be produced by any of a variety of viruses, arthropod-borne or otherwise; bacteria; fungi; protozoa; or chemicals. For reasons such as these, diagnostic dependence on clinical evidence alone has lost much of its validity in regard to infectious diseases. In the close partnership now necessary between the clinician and the laboratory diagnostician, the laboratory acts as arbiter when the identity of a causative agent is in doubt.

But the diagnostic dilemma is not necessarily solved when a disease state is correctly tagged with the family name of the causative organism. Also to be taken into consideration are the types and strains, which often have individual antigenic properties. This is true of other organisms as well. Only certain strains of the staphylococcus, for example, are antibiotic-resistant and produce epidemics.

The classification and naming of viruses is still in a fundamental stage. Cocksackie viruses were so named because they were first isolated from residents of Cocksackie, N.Y. Together with polio and ECHO viruses, they are classified as enteroviruses because their habitat is the intestinal tract. Their most drastic effect is on the central

nervous system. Members of the ECHO group were originally described as orphans in search of a disease. The word ECHO was coined from the first letter of each attribute of these viruses—enteric, cytopathic, human, and orphan, the latter in the sense that they had no known link with any pathologic condition at the time they were discovered.

Arthropod-borne viruses do not have one single property in common. Many that are transmitted by mosquitoes and ticks produce dissimilar diseases, such as encephalitis, yellow fever, dengue, and a host of others. Some have been placed in the group solely because they have an antigenic relationship with known members. Like the bat salivary gland virus, they may not be spread by arthropods at all.

Dissatisfaction with nomenclature dates back to antiquity. More than two thousand years ago, Plato commented in *The Republic*, "They do certainly give very strange and newfangled names to diseases."

Despite the number of viruses known today and the unfolding of their pathogenic role, the etiology of the vast majority of infections of the respiratory, gastrointestinal, and central nervous systems is still undetermined. The present status of virology has been compared to that attained in bacteriology fifty years ago.

Probably no infectious diseases are more persistent and widespread than those of the respiratory tract. The bacterial diseases have been studied extensively, and a great deal of progress has been made against them. Advanced chemotherapy has pulled down the mortality from these diseases, although the morbidity rate still leaves much to be desired. Tuberculosis, for example, has plummeted from first to twelfth place as a death-dealing infectious disease, but the attack rate has not dropped as drastically. Bacterial pneumonias also claim far fewer lives than they once did. The viral respiratory diseases are now of major concern. Until recently, a sound etiologic relationship of viruses to major human respiratory diseases had been established only for influenza and psittacosis. Both of these viruses were identified during the 1930's, and both have been the subject of extensive investigation.

Influenza pandemics and epidemics have long been of concern to people who are interested in respiratory diseases. Even when the influenza itself is not particularly severe, it is followed by a wave of mortality in excess of normal expectations. These deaths are usually attributed to pulmonary and cardiac conditions. It is well

known that pathogenic bacteria take advantage of the derangement of the respiratory tract during an influenza attack and readily produce pneumonia. Of the many possible complications of influenza, only the bacterial yield to chemotherapy.

Since the influenza virus was isolated in 1933, 4 major immunologic types—A, B, C, and D—have been differentiated. Most epidemics have been associated with type A, and some outbreaks, with type B. Type C appears to be relatively rare. Type D is variously known as parainfluenza 3 or Sendai virus. We will refer to it later. Each of these influenza virus types has a multitude of strains, which occur in families in types A and B. These strains are so closely related within their respective families that immunity to one confers immunity to others in the same family but does not extend to other families of the same type.

Influenza viruses of one type or another are almost constantly present in a few people everywhere, and the general population develops some degree of immunity against them. When variants develop, as they frequently do with influenza viruses, the new strains that are least readily neutralized by existing antibodies in the population have the best chance to survive and establish themselves as successful pathogens. Specific vaccines will probably always have to be developed against such strains under emergency conditions. Vaccination is our only weapon against epidemic influenza.

The Asian strain that swept across the world in 1957 was a new family of type A virus, with antigenic properties different from those of other strains. Neutralizing antibodies were found only in a few elderly people in the Netherlands and the Boston, Mass., area. Possibly the Asian strain was somehow related to the one responsible for the 1890 influenza epidemic. Because the Asian strain was different from those that have been prevalent within the lifetime of most people, susceptibility was high.

The story of the way in which the Asian strain was isolated and its spread was traced from Hong Kong around the world is a familiar one. Excellent health teamwork, national and international, played a large part in helping this country prepare for the epidemic. Time and place also worked to our advantage. Summer intervened between the alert signal and the establishment of the virus in this country. During the relatively influenza-free summer months, approximately 40 million doses of vaccine were started in production. Had the mutant strain

developed in this hemisphere north of the equator, we would not have had this period of grace.

During one week in October, according to National Health Survey estimates, almost 12 million Americans were in bed with acute upper respiratory disease. Asian influenza was accompanied by the familiar pattern of excess pneumonia-influenza mortality, which followed its successive waves, particularly among the elderly and the cardiopulmonary disabled. In the six-month period from October 1957 through March 1958, there were nearly 88,000 excess deaths in this country as compared with comparable periods in 1956 and 1957. It is observations such as this, together with the rapidity of events in fatal cases, which prompt physicians to practice routine annual influenza immunization in their cardiac and pulmonary patients and in the elderly people whom they serve.

A profusion of myxoviruses, ECHO viruses, adenoviruses, and others have been identified and associated with respiratory infections in recent years.

When the myxoviruses were first identified, the group included only the viruses for influenza, mumps, and Newcastle disease of fowls. A number of new viruses have since been added to the group. Influenza virus D, better known as Sendai virus and as parainfluenza 3, was isolated in Japan, where it was reported to be an important cause of pneumonitis in infants. It was also recovered from rodents and swine. In one city in the United States, this virus caused 50 per cent more cases of acute respiratory disease and pneumonia in hospitalized children during the Asian influenza epidemic than did the Asian strain itself. It has now been isolated from nasal secretions and diseased lungs in calves and cattle during outbreaks of respiratory disease among them. This virus is being studied by the Public Health Service and the University of Maryland as a possible cause of shipping fever in livestock. Parainfluenza virus 3 spreads so readily from one person to another that no animal source is needed to explain frequent human infections. However, the animal study may provide a better understanding of viral respiratory disease in human beings as well as in cattle and also may give some indication of the value of vaccines against it.

When adenoviruses were found in association with acute respiratory illness in military populations in 1954, it was hoped that the etiology of much undiagnosed illness would be established. Although these agents have been isolated from human tonsils and adenoids all over the world,

they account for only a small proportion of the total respiratory illness in civilian populations. During the first flush of hope about these viruses, a vaccine was developed against them. It is now used chiefly in military populations, where the incidence of adenovirus infections remains high, particularly among recruits.

Some of the ECHO 10 viruses in the enterovirus group are thought to cause respiratory infections in children. Sabin has proposed reclassifying ECHO 10 as type 1 of the new Reovirus group because of its size, effect on cells, epidemiologic pattern, and association with respiratory diseases in man and lower animals.

In quest of the cause of the common cold, investigators have recovered a number of miscellaneous viruses, but none has proved to be the single cause. The Salisbury Hospital's Common Cold Research Unit in England has persistently attempted to establish the relationship of a variety of viral isolates with common cold symptoms in human volunteers. They introduce the isolates directly into the nasal passages of the volunteers. It appears that humidity and a number of unknown factors influence the host response to infection.

Another group, the enteroviruses, is composed of at least 60 distinct strains of poliomyelitis, Coxsackie, and ECHO viruses. They have much in common in regard to laboratory characteristics, particle size, disease syndromes, and epidemiologic pattern. These viruses are important public health problems because they cause widespread epidemics, particularly during the summer. Man appears to be the natural host for these viruses, although some ECHO types have been recovered from monkeys, cattle, and swine. Transmission of the enteroviruses follows a fairly direct route from person to person.

The 3 types of polioviruses are the best known of this classification. They have been studied for decades, and an over-all picture has been obtained of the pattern of their occurrence, routes of transmission, and results of their presence. The polioviruses are the only members of the category for which we have some measure of control. The Salk vaccine has proved highly effective in preventing paralytic cases of polio and has maintained its safety since the initial incident in 1955. Despite the availability of a safe, effective vaccine, the annual case rate of poliomyelitis has continued to rise in the past several years. Analysis of morbidity data shows the heaviest concentration of paralytic cases among unvaccinated preschool children in low socioeconomic groups. Experience has not con-

firmed the belief that people universally and voluntarily will avail themselves of the protection afforded by the vaccine. Some 34 million people in the vulnerable, under-40-years-of-age group, including half of the children under 5, have not had a single dose of the vaccine.

Debates have raged around the comparative advantages of a killed virus vaccine and one made from attenuated strains that have lost their power to attack the nervous system. Proponents of the killed vaccine point to its safety. It is recognized, however, that an immunized individual can still pick up, propagate, and excrete the virulent strains. Although he himself is immune, he constitutes a hazard to his susceptible associates. Proponents of the live vaccine point to the possible extension of protection beyond the primary recipients of the vaccine. They state that secondhand immunizations occur as attenuated strains pass naturally from vaccinated individuals to their contacts. Should these naturally infected persons then become links in the further transmission of the strains, succeeding generations of the tamed and disciplined strains might gradually supplant the wild virulent strains in the population. The successful application of this theory depends on the continued stability of the attenuated strains through innumerable human passages. To operate as predicted, the strains would not only have to retain their capacity to induce antibodies against their virulent counterparts but also would have to show no sign of reverting to their neurotropic characteristics.

Attenuated vaccines have already been tested, with no apparent ill effects, in Latin America and Russia and are now being tried under carefully controlled conditions in other parts of the world, including Minnesota. In a recently completed study in a Philadelphia suburb, live attenuated poliovirus was administered orally to 18 infants. Antibody production was good, with 17 of the children responding to all 3 types of virus and 1 to types 1 and 3 only. Within fifteen weeks, 42 per cent of the siblings and 11 per cent of the susceptible adults had acquired antibodies. Live virus was then fed to the seronegative adults and to siblings under 14 years of age, with 50 per cent and 91 per cent, respectively, developing infection. Attempts to reinfect the individuals who had been fed virus or who had acquired a natural infection showed that they had developed resistance to it, for they excreted virus for only two days. There were no illnesses related to the vaccine. After 1 to 3 human passages, recovered virus was inoculated

intracerebrally in monkeys, with no significant evidence of increased neuropathogenicity.

Another study, at Tulane University School of Medicine, is under way to determine the susceptibility of newborn infants to the Sabin live poliovirus. At 2 and 30 days of age, infants will be fed various doses of all 3 types of attenuated virus, separately and in combination. Blood and fecal specimens will be studied for the presence of antibodies and for the excretion of virus, respectively.

In Minnesota, the Department of Health is carrying out controlled studies of live poliovirus vaccine based on the Cox strains. Metropolitan, rural, and suburban areas are well represented by Minneapolis, St. Paul, Duluth, the tri-county region including Meeker, Kandiyohi, and Swift Counties, and the Minneapolis suburbs of St. Louis Park and Bloomington. At this time, approximately 100,000 persons of various age and socioeconomic categories have received trivalent vaccine or placebos. Surveillance has been maintained in all areas in an effort to evaluate fully any possible adverse reactions to the vaccine. So far, there has been no suggestion of any untoward events and no report of vaccine related illness. In the Minnesota studies, the natural spread of the attenuated strains seems to be pretty well limited to family and household contacts of the vaccinated individuals.

These examples indicate the painstaking and detailed investigations that must be undertaken before a vaccine can be released for general use.

Coxsackie and ECHO viruses are more versatile than polioviruses. Their repertoire includes nonspecific febrile illness, with or without rash; aseptic meningitis; herpangina; pleurodynia; myocarditis; and, sometimes, respiratory and diarrheal diseases, depending on the species and strains involved. Clinical features of enterovirus infections frequently overlap. For this reason, laboratory diagnosis is essential in the specific diagnosis of enterovirus disease. State and regional virus laboratories across the country have access to the Communicable Disease Center's reference diagnosis service when the identity of an isolate is in doubt.

Coxsackie viruses, discovered in 1948 during poliomyelitis investigations, were found initially in association with nonparalytic polio, mainly in young people. A few years later, they were joined by the ECHO viruses.

As producers of aseptic meningitis, Coxsackie and ECHO viruses act very much like polioviruses. Paralysis, especially residual paralysis, is rare. Approximately a score of paralytic cases

have been reported in the literature during the past ten years. Development of paralysis is generally presumed to indicate poliomyelitis; absence of the syndrome does not necessarily absolve the polio agents.

Coxsackie and ECHO viruses are believed to be world-wide and, like poliovirus, more endemic in warmer climates. Infection is year-round, with more illness during warm months. They follow the poliovirus pattern in regard to age, sex, race, and socioeconomic groups.

Epidemics are typically localized and sporadic, varying in etiology from place to place during the same year. Iowa has studied the enteroviruses extensively, having had unusual experience with them. Within a radius of 100 miles, an epidemic of ECHO 4 occurred in 1955, of Coxsackie B5 in 1956, and of poliovirus 1 in 1959.

Every possible combination of enteroviral infection has been found. This has led to a great deal of speculation regarding interference, non-interference, and potentiation between virus types. So far, there is little direct information. Sabin and others have encountered instances where naturally occurring enteroviruses appeared to interfere with live virus polio vaccine. Some authors have reported that polio epidemics started later than usual when they were preceded in the area by other enteroviral epidemics.

Enteroviruses are intimately associated with man in the absence of disease, and actual illness appears to be an unusual response. Perhaps this should not be considered strange, since nature furnishes a number of instances in which human-beings, other animals, plants, insects, and even bacteria harbor viruses, yet manage to keep the host-parasite relationship in a state of equilibrium.

Because enteroviruses constitute an important public health problem, the Communicable Disease Center is initiating a surveillance program on them in 10 to 12 cities throughout the country. Monthly fecal specimens and annual blood specimens will be obtained from healthy young children for analysis of seasonal, annual, and geographic variation of polio, Coxsackie, and ECHO viruses. By comparing the subclinical flow of enterovirus infections with the occurrence of clinical disease, some clue may emerge to explain, and possibly to predict, outbreaks.

Also, the study will provide a means of accumulating a background picture of the prevalence and characteristics of naturally occurring wild polioviruses and other enteroviruses before the live virus polio vaccine is used. It should

then be possible to determine the effect of its use on the natural occurrence of these viruses.

Many of the viruses, old and new, are specific for one kind of animal or another. The arthropod-borne viruses are particularly interesting, because at least 3 types of creatures have a part in the infection cycle. The first viral disease in which arthropods were shown to be essential in maintenance and dissemination was yellow fever. Later, their role was defined in regard to dengue and several encephalitis viruses. Over 100 viral agents are now known to be transmitted by arthropods, particularly mosquitoes and ticks. In this country, the most important arbor viruses, as they are called for brevity, are eastern, western, and St. Louis encephalitis viruses. Each of these is recognized as a public health problem somewhere in the United States. Birds and mosquitoes apparently maintain eastern encephalitis in fresh water swamps in the eastern part of the country, hibernating mosquitoes are thought to be important in keeping western encephalitis alive through adverse weather in the western part of the country, and birds may carry latent St. Louis virus. Man appears to be an incidental and terminal host in these infections.

One highly specialized type of virus invades bacterial cells and literally disintegrates the host cell. These bacteriophages have been put to use by the laboratory diagnostician and the epidemiologist. Antibiotic-resistant epidemic strains of staphylococcus can be identified by phage typing. Their spread can then be traced through the hospital or community. Phage typing is of no significance in scattered infections, but it is an invaluable tool in an epidemic. A few years ago, the technic helped track down the source responsible for 34 cases of typhoid fever in 8 states. The common denominator in this outbreak was a summer meeting of a religious sect in the Midwest.

The ability of bacteriophages to destroy the bacteria susceptible to them has led to some conjecture in regard to their potential therapeutic value. So far, however, nothing positive has come out of limited experimental work.

Judging from the sheer number of viruses brought to light in recent years and the number of remaining diseases for which we have no known cause, may we not expect a growing harvest of new viruses and a link between them and these diseases? Even now, the relationship between viruses, tumors, and other diseases of unknown etiology is being probed. Also under scrutiny is the disease-producing effect of viruses

and bacteria in combination. The serious import of bacterial complications superimposed on viral infections, such as influenza, is a case in point.

As we move into a new way of life characterized by urbanization, industrial expansion, and technologic development, we need to explore their relationship with viral infections. Physical and chemical contamination of food, water, and air may have more bearing on the subject than is apparent at first glance.

For example, abundant evidence exists that viral hepatitis can be water-borne. Many communities are pouring enormous quantities of disease-carrying sewage, raw and untreated, into the water sources of their downstream neighbors. Water treatment is designed to cope with enteric bacteria, not with viruses. In addition to human and domestic wastes, some 7,000 new industrial and agricultural chemicals also are finding their way into our water supply each year. If, as some scientists surmise, detergents dumped into our water increase cell permeability, these chemicals alone may be heightening our susceptibility to hepatitis and other viral infections. What the cumulative effect of all the contaminants may be is unknown.

Air, the environmental factor that surrounds us twenty-four hours a day and without which we cannot exist, has become the carrier of a large amount of pollutants in urban areas everywhere. The cleanest air in these urban communities is 5 times as dirty as that in nonurban areas, according to the National Conference on Air Pollution held in Washington, D. C., in 1958. Automobiles, barbecue pits, trash burners, furnaces, and domestic and industrial smoke stacks belch harmful gases and unconsumed chemical particles into the air. The National Air Sampling Network monitors the air in 240 cities and non-urban sites, analyzing samples for 30 specific inorganic substances. Some of these substances are being tested for possible tumor production in experimental animal colonies. Follow-up studies of residents of Donora, Pa., and other smog-stricken cities show that contaminated air apparently plays a role in the development of chronic diseases—respiratory and chest conditions, among others. It seems quite likely that contaminants in the air pave the way for viral respiratory infections.

The use of antibiotics in animal feeds raises questions, also. If sufficient amounts are carried over into our table meats and poultry, they may decrease the normal complement of intestinal bacteria, leaving the field free from competition for the viruses.

These are only a few of the possible ways in which modern life can contribute to our susceptibility to viruses. Hovering over the entire subject, of course, is the question of host response. We still do not know what makes one person sicken and another remain well under the onslaught of pathogenic organisms. We are still speculating about the long-term effect of the stresses inherent in our way of life.

Virology may be on the threshold of its golden age, just as bacteriology was many years ago. Research is constantly broadening the horizon. While research is seeking answers to basic questions, there is much we can do on the basis of the knowledge we already have. Like the poet, we can try to see the unseen in the seen. This approach is not unrealistic, for the effects of viruses can be seen and studied although the

agents themselves are not readily visible. Pioneers in tuberculosis work tackled control programs while knowledge of the organism was in its infancy. In the viral disease field, epidemiologists and physicians are helping to trace the pattern of viral spread and behavior, whether the agents are known or unknown, single or multiple. They have ample precedent for bringing their own resourcefulness to bear on the problem of control.

The idea of viruses is intriguing. It is full of subtleties and imponderables that beckon us on. Best of all, it plants within us the virus of ideas that will take shape and grow. Where they will lead, we cannot predict. Bacteriology gave medical science the investigative approach. Virology, as it matures, may open new medical frontiers that now seem dim and faraway.

PERSONS WITH CORONARY heart disease no longer should be considered untouchable for life insurance; life expectancy of selected groups with coronary disease is far better than was formerly supposed.

Insurability usually is limited to persons not expected to exceed a long-term mortality ratio of 500 per cent, or 5 times as many deaths as would occur in the general population. The mortality ratio in the first two years after a coronary attack is 600 to 700 per cent. The ratio improves to 400 per cent in the third to fifth years, 300 per cent in the sixth to tenth years, and 150 per cent in the eleventh year.

Generally, 3 of 5 persons surviving acute myocardial infarction live another five years, 1 of 3 another ten years. Milder attacks, more complete recovery, and older age make the long-term outlook more favorable. However, when disorders predisposing to progression of coronary disease, such as diabetes, obesity, hypertension, and elevated serum cholesterol, are associated, life expectancy is shortened.

Asymptomatic electrocardiographic abnormalities also signify an adverse effect on survival. Mortality is about 3.5 times normal in persons with major T wave abnormalities and 2 times normal in those with minor T wave changes. Mortality is almost 3 times greater among persons with ischemic electrocardiographic changes after exercise than among those with negative responses. Although formerly considered an ominous finding, bundle-branch block is not accompanied by a striking increase in mortality, providing physical impairments are not associated.

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Trigger Thumb

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In trigger thumb, a snapping sensation occurs with extension and flexion of the distal phalanx of the thumb. The complaint is usually localized to the interphalangeal joint of the thumb, and, in advanced cases, the distal phalanx may be locked in partial flexion. The condition may be congenital or acquired and occurs, therefore, in children, infants, and adults. Synonyms include snapping thumb, stenosing tenovaginitis, stenosing tenosynovitis, and stenosis of the sheath of the flexor pollicis longus tendon.

SIGNS AND SYMPTOMS

Two types of trigger thumb occur, one in children and infants, in which the syndrome is congenital, and a slightly different type in adults, in which symptoms follow injury to the flexor pollicis longus tendon where it passes through its tunnel at the level of the head of the first metacarpal bone.

When the thumb of an infant or young child is involved, the abnormality is usually noted by a parent or grandparent. The distal phalanx of the thumb is held in a moderately flexed position, and, as this is a natural position of the thumb of an infant, the deformity may not be discovered for a number of months. Examination reveals that the thumb is held with the distal phalanx flexed approximately 45 degrees. Passive extension of the phalanx past this point may be impossible or may occur with a clicking sensation as the thumb extends at the interphalangeal joint. A nodule is palpable in the flexor pollicis longus tendon at the point where it crosses the head of the first metacarpal bone. The nodule is usually not tender.

In adults, it is rare to find the thumb actually locked in flexion. The usual symptom is a snapping sensation, which the patient localizes to the interphalangeal joint. Although the adult may give no history of injury to the hand, an acute or chronic type of trauma frequently precedes the onset of symptoms. One patient in this series stated that she had not injured her thumb but had peeled 100 lb. of potatoes while working in

the kitchen of a hospital a day or two before her symptoms began. Another patient's difficulty began after shoveling snow and turning a mattress on her bed.

Examination, as in children, reveals a nodule in the flexor pollicis longus tendon at the level of the first metacarpal head. As the thumb is not usually locked in flexion, this nodule is readily palpable as the patient extends and flexes the distal phalanx of the thumb. A moderate amount of pain frequently occurs as the nodule in the tendon passes through the stenosed tendon sheath. Tenderness is usually present over the nodule.

DIFFERENTIAL DIAGNOSIS

In children whose thumbs are held flexed at the interphalangeal joint, congenital contracture of the thumb may be confused with trigger thumb. Although differentiation may be difficult, the presence of the enlarged nodule at the level of the metacarpal head is helpful in establishing the diagnosis.

Chronic dislocation of the interphalangeal joint of the thumb may occur both in infants and in adults and is diagnosed by x-ray examination.

Ankylosis of the interphalangeal joint may occur and may be distinguished from trigger thumb by the presence of a few degrees of passive motion in flexion when the pathology lies within the flexor pollicis longus tendon and sheath.

ETIOLOGY

Stenosing tenosynovitis in adults is due to acute or minor repeated traumata to the flexor pollicis longus tendon and its sheath, producing inflammatory changes which are followed by scarring of the tendon sheath and stenosis and formation of a nodule within the substance of the tendon. Fahey and Bollinger¹ have shown that degenerative changes and fibrous tissue proliferation occur within the fibrous sheath of the tendon. In one of their cases, a ganglion was present on the volar portion of the tendon sheath. In case 3 of the present series, a small ganglion was attached to the flexor pollicis longus tendon adjacent to

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the nodule. Fahey and Bollinger noted that the tendon sheath was usually markedly thickened, sometimes 2 to 3 times its normal thickness.

When the condition occurs in children, the etiology is less clear. In reporting 7 cases, White and Jensen² noted that 2 of their patients had a familial history of the disease. They postulated a congenital abnormality in the anatomic relationship of the flexor pollicis longus tendon to the lateral head of the flexor pollicis brevis and to the adductor pollicis. They suggested the possibility of a band between these muscles passing over the tendon sheath of the flexor pollicis longus as a possible factor in producing stenosis. However, at the time of surgery, they were unable to substantiate the presence of such a band.

Possibly trauma is a factor in the production of stenosing tenosynovitis of the long flexor of the thumb in children as well as in adults. Pressure may be exerted on the tendon and its sheath by compression against the head of the first metacarpal due to the infant's thumb being clenched in flexion by his closed fist.

TREATMENT

Surgical treatment for trigger thumb is both simple and uniformly effective. A short longitudinal incision is made over the volar aspect of the head of the first metacarpal, and the sheath of the flexor pollicis longus tendon is exposed by blunt dissection. The sheath is opened longitudinally through its thickened portion, and the tendon is inspected. Typically, a nodule is present in the tendon which is associated with the constriction in the sheath. The sheath is sufficiently opened both distally and proximally to allow free motion of the tendon as the distal phalanx of the thumb is flexed and extended. The nodule in the tendon is not disturbed. The wound is closed with interrupted catgut sutures through the subcutaneous tissue and silk skin sutures. No immobilization is used, and the patient is encouraged to begin mild active motion of the thumb as soon as he recovers from his anesthetic.

Other authors have advised additional measures at the time of surgery. Fahey and Bollinger¹ carry out local excision of the flexor tendon sheath and note that bowstringing does not occur following this procedure. Turek³ believes that the flexor pollicis longus tendon and its sheath are compressed not only against the head of the first metacarpal but also against the sesamoid bones in the heads of insertion of the flexor pollicis brevis. In adults, he advocates incision of the constricted tendon sheath both medially and laterally and removal of the sesamoids as well.

All cases in our series responded satisfactorily

to simple incision of the flexor pollicis longus tendon sheath.

CASE REPORTS

Case 1. S.R., aged 8 months, was seen in the office August 22, 1949. One week previously, the boy's mother had noted that the distal phalanx of the right thumb was held in a flexed position. She tried extending the thumb passively and was able to obtain extension but felt a snap in the thumb as extension occurred. She tried to tape the thumb in extension, but the tape came off ten minutes later and the deformity recurred.

Examination revealed that the right thumb was held flexed at the interphalangeal joint. The child's mother applied force to the thumb, and the phalanx extended and remained in this position. A small nodule was palpable in the flexor pollicis longus tendon at the metacarpal phalangeal joint. X-ray examination of the hand was negative.

The child was operated upon August 26, 1949. The sheath of the flexor pollicis longus was found constricted at the level of the metacarpal head. The sheath was incised longitudinally along its lateral border. The tendon was inspected, and a small nodule was found.

The patient was last seen October 13, 1949. Motion in the thumb was normal.

Case 2. J.E., a 3-year-old girl was in the office September 29, 1950, because of limited motion of her left thumb. Four months previously, the child's grandmother had noted that the youngster was unable to completely extend the distal phalanx of the thumb. The child's mother volunteered that the child had always held her hands clenched with the thumbs inside of the palms.

Examination revealed that the thumb was held flexed approximately 45 degrees at the interphalangeal joint. A nodule was palpable in the flexor pollicis longus tendon at the level of the first metacarpal head. Passive movement of the distal phalanx was from 90 to approximately 135 degrees, at which point resistance was present to further extension. X-ray examination of both hands was negative.

Operation was carried out on October 27, 1950. The sheath of the flexor pollicis longus tendon was exposed and was incised longitudinally. A nodule was noted in the tendon at the level of the first metacarpal head. As soon as the sheath was opened, the tendon was observed to glide freely, allowing normal motion of the distal phalanx of the thumb.

The child was last seen November 9, 1950, at which time flexion and extension of the left thumb were normal.

Case 3. Mrs. H.H., aged 54, was seen in the office September 27, 1951, complaining of a snapping sensation of the left thumb of 3 months' duration. The patient stated that she had sustained no injury to the hand but had noticed snapping in the interphalangeal joint of the left thumb and was unable to completely extend the distal phalanx of the thumb.

Examination revealed a palpable click over the palmar aspect of the first metacarpal phalangeal joint with extension and flexion of the thumb. Active extension of the distal phalanx was limited approximately 30 degrees. Roentgenogram of the hand was normal.

The patient was operated upon October 13, 1951. The sheath of the flexor pollicis longus tendon was exposed at the level of the first metacarpal head. A small ganglion measuring approximately 4 mm. in diameter was found attached to the flexor pollicis longus tendon at the

level of the metacarpal head. The tendon was thickened and enlarged in the area of attachment of the ganglion. The tendon sheath was incised along its lateral aspect, and the ganglion was removed.

The patient was last seen February 27, 1952, at which time motion in the interphalangeal joint was from 180 to 120 degrees. She stated that the catching sensation in the thumb had disappeared and she was able to crochet.

Case 4. H.T., a 4-year-old boy, was seen in the office January 14, 1954. His mother stated that he had been unable to extend the distal phalanx of the right thumb for approximately two years. The disability was noted after the patient caught the thumb in an oven door.

Examination revealed a palpable nodule in the flexor tendon of the right thumb at the level of the first metacarpal head. Motion in the distal phalanx was normal, but a palpable click was present over the first metacarpal head with extension and flexion of the distal phalanx of the thumb. There was moderate tenderness over the flexor pollicis longus tendon in the region of the nodule. Roentgenogram of the hand was normal.

The patient was operated upon February 3, 1954. The tendon sheath of the flexor pollicis longus tendon was found to be stenosed at the level of the first metacarpal head. The sheath was opened on its lateral aspect, and the tendon was inspected. A small nodule was present in the tendon. Following incision of the sheath, motion in the thumb was normal.

The patient was last seen February 25, 1954, at which time he could move his thumb normally.

Case 5. L.H., a 2-year-old boy, was seen in the office June 19, 1956, because of a deformity of both thumbs which had been noted by the child's mother two months previously. The mother stated that the child was unable to completely extend the thumbs at the distal joint and she had noted a hump at the level of the first metacarpal head in the flexor pollicis longus tendon.

Examination revealed that extension of the distal phalanges of the thumbs was limited at 150 degrees on the right and 160 degrees on the left. Flexion of both thumbs was normal. Nodules were palpable in the flexor tendons at the level of the heads of the first metacarpals. Roentgenograms of both hands and wrists were normal.

The child was operated upon July 10, 1956. The tendon sheath of the flexor pollicis longus of each thumb was incised on its radial aspect, and the tendon was inspected. A small nodule was present in the tendon bilaterally.

The child was last seen in the office January 25, 1957, at which time motion was normal in both thumbs.

Case 6. H.L., a 54-year-old woman, was seen January 2, 1957, complaining of a tender mass at the base of the right thumb of two months' duration. The patient stated that the thumb had not been injured.

Examination revealed a palpable nodule in the flexor tendon of the right thumb at the level of the metacarpal phalangeal joint. Extension in the distal phalanx was normal, but flexion was limited approximately 45 degrees. Roentgenogram of the hand was normal.

The patient was operated upon February 20, 1957. The tendon sheath of the flexor pollicis longus tendon was exposed at the level of the first metacarpal head. The nodule in the tendon was palpable and visible through the tendon sheath and was seen to move with extension and flexion of the distal phalanx of the thumb. The tendon sheath was incised laterally, following which motion was normal in the distal phalanx of the thumb.

The patient was last seen March 4, 1957, at which time motion in the right thumb was normal.

Case 7. O.J., a 67-year-old man, was seen June 17, 1957, complaining of a catching sensation in the left thumb of one month's duration. The patient stated that this began after he twisted his thumb in bed one night. The previous day he had been using his hand excessively picking rocks from a field on his farm.

Examination revealed tenderness over the volar aspect of the first metacarpal phalangeal joint of the left hand. Extension and flexion of the distal phalanx of the thumb revealed a palpable click over the head of the first metacarpal. A roentgenogram was made of the hand and was negative.

The patient was operated upon June 22, 1957. The tendon sheath of the flexor pollicis longus tendon was exposed over the head of the first metacarpal and incised longitudinally. A swelling was present in the tendon at the level of the first metacarpal head.

The patient was last seen in the office July 5, 1957, at which time he had normal use of his left thumb.

Case 8. G.S., a 3½-month-old girl, was seen October 18, 1957, because she had been unable to extend her right thumb since birth.

Examination revealed a palpable nodule at the level of the first metacarpal head in the flexor pollicis longus tendon. Extension of the distal phalanx of the thumb was limited approximately 45 degrees.

A roentgenogram was made of the right hand and was normal.

The child was operated upon October 24, 1957. The tendon sheath of the flexor pollicis longus tendon was exposed at the level of the first metacarpal head and incised longitudinally. The tendon was inspected, and a small nodule was found at the level of the metacarpal head. Following incision of the sheath, the thumb extended and flexed normally at the interphalangeal joint.

The patient was last seen October 30, 1957, at which time motion in the right thumb was normal.

Case 9. C.Z., a 15-year-old girl, was seen November 27, 1957, with a complaint of inability to extend the distal phalanx of the right thumb of fourteen years' duration.

Examination revealed that she was unable to extend the distal phalanx of the thumb actively past 150 degrees. Application of passive force to the distal phalanx produced complete extension with a palpable click at the first metacarpal phalangeal joint. A roentgenogram of the wrist and hand was normal.

The girl was operated upon May 26, 1958. The flexor pollicis longus tendon sheath was exposed at the level of the first metacarpal head and incised longitudinally. The sheath appeared constricted at the level of the metacarpal head, and the tendon proximal to the constriction was enlarged. Following incision of the sheath, motion in the thumb was normal.

The patient was last seen June 19, 1958, at which time she was able to move her thumb normally.

Case 10. Mrs. E.S., 63 years of age, was seen April 29, 1958, complaining of inability to extend the distal phalanx of the right thumb of two weeks' duration. The patient stated that she had not injured her thumb but had peeled 100 lb. of potatoes at the hospital at which she worked a day or two before her symptoms began.

Examination revealed a tender nodule palpable over the volar aspect of the first metacarpal head of the right hand. Motion was normal in the distal phalanx of the

thumb, but a palpable click was present over the metacarpal head with extension and flexion.

The patient was operated upon May 27, 1958, and the flexor tendon sheath of the flexor pollicis longus tendon was exposed at the level of the first metacarpal head. The sheath was incised longitudinally. A nodule was present in the tendon at the level of the metacarpal head. Motion in the thumb was normal after incision of the sheath.

The patient was last seen June 13, 1958, at which time motion in the right thumb was normal.

Case 11. Mrs. E.S., a 58-year-old housewife, was seen first on May 6, 1959, because of a catching sensation in the right thumb of six to seven weeks' duration. The patient stated that the difficulty began after shoveling snow and turning a mattress on her bed.

Examination of the right hand revealed a tender nodule in the flexor pollicis longus tendon at the level of the head of the first metacarpal. Extension and flexion of the distal phalanx of the thumb were normal, but a palpable click was noted in the region of the metacarpal phalangeal joint. A roentgenogram of the hand was normal.

The patient was operated upon May 13, 1959. The tendon sheath of the flexor pollicis longus tendon was exposed and was found to be thickened at the level of the metacarpal head. The sheath was incised along its lateral aspect, and the tendon was inspected. A nodule was present in the tendon proximal to the stenosed area in the sheath. Motion was normal in the thumb after incision of the sheath.

The patient was last seen May 26, 1959, at which time she had normal motion in her thumb.

Case 12. E.B., a 7-year-old boy, was seen June 2, 1959, with a flexion deformity of both thumbs which had first been noted at the age of 3 years.

Examination revealed that both thumbs were held flexed at the interphalangeal joints. Passive stretching of the distal phalanges failed to produce extension of the thumbs. Palpable nodules were present over the palmar aspects of the first metacarpal phalangeal joints.

Roentgenograms of both hands were normal.

The patient was operated upon June 3, 1959. The sheaths of the flexor pollicis longus tendons were exposed at the level of the first metacarpal heads. Both sheaths were thickened and stenosed, and, when opened, nodules were found in the flexor pollicis longus tendons. Motion was normal in the thumbs after incision of the tendon sheaths.

The patient was last seen July 31, 1959, at which time the range of motion in the interphalangeal joints of both thumbs was normal.

SUMMARY

The syndrome of trigger thumb is discussed. Two types exist, one in children and infants, which is congenital, and the other in adults, which usually follows acute or chronic injury.

Twelve case reports are presented. The age at the time the patients were first seen ranged from 3½ months to 67 years. Five of these patients were adults, and 7 were children 15 years of age or younger. The right hand was involved in 7 instances, the left in 3, and both hands in 2. Five of the patients were male and seven were female.

Surgical treatment is uniformly effective and consists of incision of the constricted portion of the sheath of the involved flexor pollicis longus tendon.

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WITH A FEW exceptions, long limb bones and ribs are denser than vertebrae, cervical vertebrae are denser than other segments of the spinal column in men but not in women, bones of the male skeleton are denser than those of the female skeleton, and bones of the Negro skeleton are denser than those of the white skeleton. Within each sex or race group, bones decrease in density with age at a uniform and parallel rate.

These data were obtained by studying the skeletons from 40 American white and 40 American Negro cadavers, each racial group consisting of 20 men and 20 women. Age range was 25 to 100 years. Bone density was considered to be an expression of the weight of the bone per unit volume. Bones were weighed in a dry, fat-free state; volumes were measured by displacement of millet seed.

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A Survey of Electrocardiographic Practices in the Upper Midwest

REUBEN BERMAN, M.D., AND ERNST SIMONSON, M.D.

IN 1951, a survey of electrocardiographic practices in hospitals of the Upper Midwest was published in *THE JOURNAL-LANCET*.¹ Questionnaires were sent to 222 hospitals in Minnesota and the Dakotas, and 75 (34.4%) responses were received. A small group (5.3%) still adhered to the routine use of 3 limb leads; 9 hospital staffs (12%) were using 5 or 6 chest leads. However, at that time, the mode (52%) was 3 limb leads and 1 chest lead (usually 4F).¹

The hospitals of these 3 states were again surveyed by questionnaire in March 1960. The number has grown in nine years from 222 to 325.² A total of 166 replies were received from 325 hospitals, representing a 51% response. The questionnaire asked for the size and location of the hospital, the type of electrocardiographic instruments used, leads used routinely, and other leads used occasionally. Information was requested also concerning the use of lead III and aVF in deep inspiration and the availability of stress tests of various types. The table indi-

cates the results according to size of hospital. Since the survey included both rural and metropolitan centers and the total number of replies was large, it is likely that these findings may be generalized for the entire United States.

An impressive unanimity has been achieved in this region in hospitals of all sizes, with 91.6% routinely using 12 or more leads. More than 6 chest leads were used in 23 (13.8%), 14 utilizing additional leads to the left of V6 and 9 to the right of V1; 9 hospitals (5.2%) have available an esophageal lead.

The only stress test that is widely used in this area is some form of exercise, reported by 116 (69.9%). The meal test^{3,4} is used by 6 and the anoxia test by 2. Of the large hospitals with 100 or more beds, 90.6% reported using stress tests. The machine used is one of the direct writers in all of the medium size hospitals reporting and in 97% of all the hospitals.

It is evident that a minimum of 12 leads including 3 standard, 3 augmented unipolar limb, and 6 chest leads has received universal acceptance in the Upper Midwest. This represents a change in the last nine years. In this period, the chest leads have become universally accepted among hospitals large and small. The augmented unipolar limb leads also have achieved general usage.⁵ It is likely that a part of the preponderant agreement on the leads used depends upon

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ELECTROCARDIOGRAPHIC TECHNIQUES IN UPPER MIDWEST HOSPITALS

Number of beds	Total number	Direct writer	Number using 12-lead machine (or more)	Additional leads			Deep inspiration (III or aVF)	Exercise test
				Esophageal	Right of V1	Left of V6		
1 to 49	73 100%	70 95.9%	67 91.8%	0	1	3	17 23.3%	37 50.7%
50 to 99	23 100%	23 100.0%	22 95.6%	0	2	5	14 60.9%	16 69.6%
Over 100	64 100%	63 98.4%	58 90.6%	8	6	6	24 37.5%	58 90.6%
Unknown	6	5	5	1	0	0	3	5
Total	166 100%	161 97.0%	152 91.6%	9	9	14	58 34.9%	116 69.9%

the manufacturers of direct writers who incorporate a single lead switch enabling the operator to inscribe leads I, II, III, aVR, and aVL and the V leads serially by the turn of a single knob. Only 1 hospital over 50 beds and 3 small hospitals still use string galvanometers. Direct writers of various types have supplanted the string instruments recording on photographic film.

The use of ancillary leads increases with the size of the hospital. Esophageal leads and leads to the right and left of VI-V6 are used in about 10% of the large hospitals. The inclusion of lead III or aVF in held inspiration in 34.9% of reporting hospitals indicates that this facet or electrocardiography still enjoys considerable vogue.

A question on the use of vector cardiography drew affirmative answers from 2 hospitals, both research and teaching institutions where this method is being used experimentally.

We wish to thank the staff of *The Journal-Lancet* for cooperation in preparation of the questionnaire and Dr. Mitsui Okajima for his statistical assistance.

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BREATH-HOLDING TIME is an indication of cardiac status and may be used to differentiate functional from organic cardiovascular disease. Patients are instructed to inhale deeply, exhale completely, and, after a second deep inhalation, hold the breath as long as possible, exhaling at the limit of tolerance. Values are about forty-eight seconds for normal subjects and patients with class 1 cardiovascular disease and thirty-five seconds for patients with class 3 heart disease. Findings for most patients with class 2 cardiovascular disease were between these results. Tolerance of patients with pulmonary emphysema without heart failure was about twenty-three seconds and of those with functional disease, fifteen seconds. If the patient is able to walk into the examining room, breath-holding ability of twenty seconds or less suggests that symptoms are mainly functional.

R. M. KOHN: Breath-holding time in the evaluation of organic and functional heart disease. *Circulation* 20:721-722, 1959.

THUMPING THE PRECORDIUM with the fist restores heartbeat after ventricular standstill. While an electric apparatus or chemical stimulants are being readied, repeated chest blows, combined with mouth-to-mouth breathing if necessary, maintain life. In patients succumbing to intrinsic or extracardiac disease, automatism in the ventricular muscle may be reawakened repeatedly by the maneuver, the response varying with the degree of myocardial deterioration.

In the normal heart, a single ectopic beat appears with each blow. In the damaged myocardium, at first a single ectopic beat is elicited with a blow; with progressive hypoxia, a chain of ectopic beats appears, ventricular complexes widen, and ventricular flutter occurs.

A blow to the chest, like any impulse reaching the heart during a vulnerable phase, may lead to ventricular fibrillation. However, the implications of standstill are so grave that the risk of fibrillation must be accepted. The sooner blows are applied, the better the condition of the myocardium and the more likely that a single blow will elicit a single ectopic beat.

Precordial thumping should be tried even if ventricular fibrillation is suspected. Ventricular fibrillation may be confused with multiple variform ventricular extrasystoles in the electrocardiogram, or standstill may occur after fibrillation.

D. SCHERF and C. BORNEMANN: Thumping of the precordium in ventricular standstill. *Am. J. Cardiol.* 5:30-40, 1960.



Notes from a Medical Journey

Fermo (Marche), Italy
7 April 1960

Dear Jay:

Letters intended from several medical journeys remain unwritten-- days too crowded with work, evenings appropriated by gracious but demanding hosts, and nights overborne by fatigue or indolence. The time slips by on this trip too, but here at Fermo, overlooking the Adriatic sparkling 4 miles away and 1500 feet below, I am free until time to drive 20 miles to lunch with the research team at Montegiorgio, a still smaller town on a still higher hill. From my balcony, I can see part of the road, a white thread twisting up and down hills between a patchwork of fields of varied greens and the brown of newly ploughed earth. Beyond Montegiorgio, the snow-covered Sibilline Mountains stand sharply etched against the blue of the morning sky and all between sea and mountains are the smiling hills of Marche.

Here the Sabines came over two thousand years ago, fleeing from the Romans through the passes of the Appenines, building their hilltop fortresses against another rape and preying, no doubt, on the simple folk who were here before them. But soon they were all assimilated within the Empire, their levies marched with the Roman legions, and from Ancona the Roman navy set out to conquer Dalmatia across the Adriatic. Today, the great triumphal arch marking their success still stands on the water's edge at Ancona, and a vast remnant of the Roman city they built on the opposite shore forms the heart of Split, which the Italians call Spallato.

This is a region of small farms, each laid out in 5 sectors of different crops tended with loving care by all the family; 10 or 12 acres yield an astonishing produce, and both the people and their pairs of white oxen are sleek. The houses and villages are immaculate, the people are friendly and gay, the food is varied and plentiful, and only money is lacking. Wheat, sugar beets, and a few pigs and calves go to market. With the proceeds, they buy materials for clothing and farming implements and pay taxes and the "Mutua," which covers medical care. Somewhere, too, there must be enough to contribute to the Church and buy household furnish-

ings for the children when they marry. But, as in all the world today, some of the young people are not content, and more and more of them are leaving the farms.

Montegiorgio is a large agricultural village typical of a region where life is stable in spite of the migration of some of the youngsters, where there is no hunger, no serious problem of infectious disease, where contrasts between rich and poor are relatively small, and where we are sure of cordial cooperation now and in the five-year follow-up we plan. We want detailed medical data on all men aged 40 to 59 in the selected area. We cannot hope to examine every one of the 750 men on the roster, but the coverage should equal the 98% mark achieved in the study just finished at Crevalcore (near Modena).

Local doctors are helpful, but the real work is done by the team organized with the aid of Dr. Vittorio Puddu of Rome and Dr. Arrigo Poppi of Bologna. Colleagues from our research teams in Yugoslavia, Finland, and Greece come here for various periods to assure adherence to the standards adopted for all the areas in this international research program. Dr. Ivan Mohaček of Zagreb has been a constant member of the team both here and at Crevalcore and will come to Minnesota for the summer. Dr. Henry Blackburn of our staff in Minnesota spent a month at the outset at Crevalcore, and the good effects of his teaching are evident, especially in the objective recording of clinical and electrocardiographic findings.

This work, as in our other studies, is concentrated on relationships between the mode of life and the development of heart disease, especially coronary or "ischemic" heart disease, and how these may apply to prevention. We are concerned with cause and effect of course, but I have long since given over arguing about final causes. At present, it is enough to discover the sequence of events from health to disease and to be able to predict the statistical outcome in frequency of disease from the mode of life and the characteristics of the population in health. Already much has been learned.

From the data on the customary diet, we can make a fair estimate of the average concentration of cholesterol in the blood. Tell us the average concentration of cholesterol in the blood in the population, and it seems that we can predict, at least roughly, the relative frequency of coronary heart disease in the men of middle age. It is still pretty crude; the contributions of obesity, blood pressure, and activity are obscure, and the predictions are not much good for individuals. But, looking back a few years, the progress is impressive. Now we need much larger population groups in follow-up studies to be able to sort out details with more accuracy. This is a main feature of the programs at Crevalcore and at Montegiorgio; together, they cover about 1,500 men, aged 40 to 59 at the outset, who will be followed. Add the 600 Italians at Nicotera with whom we started this long-range program several years ago, the 1,500 men in rural Finland examined last year, the 1,500 in Yugoslavia studied in 1958, the 650 Greeks on the Island of Crete, and the 1,000 Netherlands soon to be studied at Zutphen, and we have nearly 7,000 men in Europe under surveillance.

Coverage in the United States is not nearly so good; in all surveys, many men refuse to cooperate and others are lost sight of because of changes in residence and occupation. But Dr. Henry Taylor now has nearly 3,000 railroad employees fully examined and for whom we think the prospect of follow-up is exceptionally good -- for the U. S., that is. The U. S. group provides one satisfaction: the "yield" of coronary heart disease so far is far richer than in any of the other groups.

Yesterday, we checked the local hospital at Montegiorgio and were impressed by both staff and facilities. About 80 of the 100 beds were occupied, and most of those were surgical; no coronary patients were to be found, and the Chief of Staff was hard put to remember when he had seen an infarct or a real case of disabling angina pectoris. This is typical of our experience in the rural areas in this part of the world. The local doctors know something about coronary heart disease because, several times a year, they go to medical meetings where coronary heart disease is a popular subject of discussion.

How much the diet has to do with the lack of coronaries here I should not like to say, though we already know that the average serum cholesterol is low. The dietary work has far to go, but certainly the usual diet here is calorically abundant and low in saturated fats. Dr. Flaminio Fidanza, who leads the Italian team, promises detailed data soon. We like the diet with its excellent bread, wonderful fresh vegetables and fruits, low-fat fresh cheese (ricotta, mozzarella, fior di latte), light, dry white wine ("verdicchio"), and meat and fish in small amounts but fine in flavor. Good soups and all kinds of pasta -- spaghetti, lasagna, fettuccini, etc. -- are a part of every main meal, of course.

Writing about food reminds me that Margaret and I should get on the road soon to be in time for lunch at Montegiorgio. She is busy on the Italian edition of our book, EAT WELL AND STAY WELL. The Finnish edition, which is well reviewed in Finland, was much easier. Incidentally, quite a few copies are being sent to Finns in Minnesota.

Anyway, it is time to think about food; we had only coffee and hot milk for breakfast, and that was five hours ago. Today, we shall lunch in the open air with blossoms overhead and wild flowers all about. Then we shall sit for a bit in the warm sun, lazily contemplating the rolling countryside, before getting back to work. The afternoon is long enough because we never dine until after eight.

With all good wishes to you and our friends at home,

As ever,



AK:ml



Hal Downey, Ph.D.

1877-1959

LEMEN J. WELLS, Ph.D. and
C. A. McKINLAY, M.D.,
Minneapolis

HAL DOWNEY was one of the foremost hematologists of our age. Soon after his death on January 9, 1959, at 81, a colleague, E. A. Boyden, wrote from Seattle, "This is the day when a loyal friend and great student of anatomy is laid to rest. . . . We will not see his like again, but we can still be inspired by his devotion to the things we hold dear and above price."

Professor Downey trained hundreds of students, who now carry on his work all over the world. He regarded the search for truth as more important than the possession of it, and he continued that search until a few days before his death. His last scientific paper reports his attempt to produce cancer cells in rats by means of tobacco tar.

He was born in State College, Pennsylvania, on October 4, 1877. His father was a mathematician who served as dean of the Academic College, University of Minnesota. His mother was a singer. As a boy, he studied for six years in Hannover, Germany. As soon as he had become fluent in German, he was admitted to the Hannover Realgymnasium. Since his teachers were all men with Ph.D. degrees, his early experience must have been a major factor in his cultural and professional growth.

This tribute, prepared by Lemen J. Wells and C. A. McKinley, is an expansion of one that was written by Lemen J. Wells and R. Dorothy Sundberg for the American Association of Anatomists (Anat. Rec. 134:272, 1959).

Back in Minneapolis, he finished high school in one and a half years. He enlisted for the Spanish-American War and served in the Philippines in Co. A of the thirteenth Minnesota Volunteer Infantry from 1898 to 1899. He was graduated from the University of Minnesota in 1903 and was granted the Master's degree in Zoology in 1904.

His student days were full of activity. His physics notebook shows that he was secretary of the Nicollet Bicycle Club but not that he was once arrested by a Minneapolis policeman for having "pedaled his racing bike too fast in town, with head down!" On many a Saturday night, he and student friends walked some 3 miles to Schiek's Cafe for conversation and "ein Glas Bier."

In 1905, he married Iva Clare Mitchell, whom he had met at the University. Her gifts included drama and creative writing. She appreciated his unique talents and always endeavored to give him ample time for his scientific work. Their three children, Phyllis, Richard, and Jean, were graduated from the University of Minnesota—Phyllis as a medical technologist, Richard as an electrical engineer, and Jean as a home economist.

His interest in hematology grew out of his study of the urogenital organs of a Mississippi River fish with many primitive characteristics, the spoon-bill sturgeon, *Polyodon spathula*. He found that the kidney is the chief hematopoietic organ in this species.

He and Mrs. Downey lived and studied in Ger-



Hal Downey, left front, member of a team of four at the dissecting table in a class in human anatomy. Photo prepared about 1905.

many in 1910 and 1911. He was in Pappenheim's laboratory at the University of Berlin in 1910 and with Weidenreich at the Anatomisches Institute in Strassburg in 1911.

Except for this period abroad, he was an active member of the staff at the University of Minnesota from 1903 to 1946. He was professor of Zoology until 1929, when he became professor of Anatomy. After his retirement in 1946, he lectured for two years at the Mayo Clinic and Mayo Foundation. Thereafter, he kept busy in his office in the Department of Anatomy up to the Christmas holidays of 1958 and 1959.

From 1913 to 1959, except during World Wars I and II, he served as American editor of the *Folia Haematologica*, published in Leipzig. In this connection, the first page of the *Minnesota Alumni Weekly* for January 27, 1913, had his portrait and a single article entitled "Professor Hal Downey, Ph.D., Honors the University and Himself." It reads:

"Dr. Hal Downey, '03, assistant professor of comparative histology, has done some remarkably good work in his special line of investigation—namely the structure of the cells in the blood and their relationship to each other, and the lymph glands. These investigations are of great importance in connection with problems of medicine. Professor Downey's work in this line is of the best that has been done in this country and brings to the department of Animal Biology, in this institution, a prestige that means much to those who are working in the field of blood investigations, and adds to the standing of the University as an institution that is doing things worth while.

"The work of Professor Downey has been recog-

nized by his selection as a collaborating editor of the *Folia Haematologica*—the international magazine devoted to investigations in the blood. Dr. Downey has also been named as the American representative of this publication to review and prepare abstracts of American articles upon blood which appear in other publications.

"Dr. Downey is the first American, as well as the first English-speaking scientist, who has ever been honored by being chosen to collaborate with Weidenreich in the preparation of an article for publication. A recent number of *Folia Haematologica* contains a review of such an article upon "Formation of lymphocytes in lymph glands and the spleen."

"Weidenreich, the great authority upon the blood, is planning a comprehensive publication upon the blood and lymph. Each section devoted to a particular field is to be prepared by an acknowledged authority in his particular field. Professor Downey is the only American who has been asked to prepare a section for this publication and he is to prepare the article upon lymph nodes. This is a very gratifying recognition of Professor Downey's standing among research students in this field.

"Dr. Downey, and the department which has given him his training, have brought to the University an honor and a distinction of which the alumni may justly feel proud."

Dr. Downey's scientific publications include many definitive contributions, abstracts, and critical reviews. He edited a *Handbook of Hematology* in 1938. This handbook has 34 contributors and 3,136 pages, in 4 volumes. He contributed chapter 16, "Monocytic Leucemia and Leucemic Reticulo-endotheliosis," and chapter 25, "The Myeloblast." In the publisher's announcement of the handbook one reads,

"No comparable work in hematology has hitherto been projected in any language" (H. E. Jordan, University of Virginia), and "I should be most enthusiastic over anything that Hal Downey published" (John F. Fulton, Yale University).

He received many honors. The regents of the University of Minnesota granted him the Outstanding Achievement Award in 1951. The Minnesota Chapter of the Society of Sigma Xi gave him its Distinguished Service Award in 1957:

"You have given this University fame in hematology. . . . Although your world-renowned paper on infectious mononucleosis describing the cells which now bear your name is known for its correlation of critical morphologic detail with clinical conditions, many other prophetic contributions on reticular and lymphatic tissues as well as on all types of blood cells are the foundations for much active research. . . . Your control of the world's bibliography of hematology . . . your editorship of the *Folia Haematologica* and your own *Handbook of Hematology* further affirm your scholarly desire to integrate ideas."

The Durban Medical School of the University of Natal, South Africa, established the Hal Downey Laboratory of Hematology in 1956. Katsuji Kato's *Atlas of Hematology*, in 2 volumes, 1954, is "respectfully and affectionately dedicated to Hal Downey." Professor Downey was an honorary fellow of the International Society of Hematology and of the European Society of Hematology.

The following 4 paragraphs are from the pen of a clinical colleague, Dr. C. A. McKinlay, who participated in the pioneering studies of infectious mononucleosis.

"The first contact of the writer with Dr. Downey was in consideration of a case seen in 1921 in which acute leukemia was the primary clinical impression, due in part to hemorrhagic tendency, marked adenopathy and lymphocytosis. The benign hematological features, however, were recognized and recorded by Dr. Downey as the cells were all mature lymphocytes.

"Interest was stirred in the problems presented by this case, which represented a new entity to clinical observers, and which was confirmed as such hematologically by Dr. Downey in a series of 9 cases reported jointly in 1923. His colored plate and description of type I, II, and III cells were classical for finer hematologic features and became in the succeeding years standard laboratory criteria for confirmation of clinical diagnosis of infectious mononucleosis.

"Although infectious mononucleosis has been recognized as a widespread disease of young people affecting practically all organ systems and although the positive heterophil test has become the common laboratory confirmation of diagnosis, the cytology as described by Dr. Downey remains, at least for the hematologist and those who can appreciate morphology, the best criterion for recognition of the disease.

"Dr. Downey will always be remembered as a

patient and diligent seeker of truth, a true scientist who gave generously of his skills and attainments to students and physicians who sought his scholarly aid."

The following 5 paragraphs are from the pen of Dr. R. Dorothy Sundberg (Wells and Sundberg: *Anat. Rec.* 134:272, 1959).

"The world will find knowledge, data, the earliest experiments in many of the morphologic phases of hematology, incomparable reviews of literature, and superb illustrations in Dr. Downey's publications. Those who studied under him, including hundreds of freshman medical students of histology, will recall that his lectures, even upon seemingly static subjects, were philosophic and inquiring. He did not carry lecture notes; he thought checking attendance an insult to both student and professor. His students in hematology would attend his lectures on the same subject for many consecutive years, if possible, for something new always was added.

"He loved best to teach in the laboratory, but here he would let the student flounder for just the appropriate length of time—a pipeful—because he firmly believed that the student would profit more from his initial struggle for understanding than from a rapid clarification of the problem.

"The world will correctly say that he was a scholar, but it is doubtful that the world can ever find in Dr. Downey's publications the essence of the man. Perhaps that essence was gentleness, personal humility, intellectuality, humor, and a certain horror of poor histologic technique—"histologic garbage"—and slipshod scientific contributions of any type. His alert and sparkling brown eyes almost always betrayed a quiet amusement with, as well as a sincere concern for, the activities of cells or people. He never forgot what either did. His students were his colleagues, not his underlings; he worked for them, though they did not know it . . .

"Dr. Downey began his work in hematology in Minnesota when many still thought platelets were parasites on red cells. This he often admitted with a grin, which indicated that he had managed to discover and teach a few things, but he more often confessed that once he forgot about malarial parasites and missed them in a blood film. Another consoling and human confession was the one he generally offered his doctoral candidates on the eve of their dreaded preliminary examinations. He claimed that he had been asked at his own doctoral examination to name the cells of the blood. He had been incensed at the question, but he found he had to be asked to name them several times before he 'got around to remembering the red cells.'

"If he could comment upon what we have written here, he would probably smile modestly and offer some clever anecdote designed to shift attention from himself."

Attention will never shift from Hal Downey. His memory will hold, his contributions to knowledge will stand through time, and his students will carry on his work. These glories we remember at each moment of sorely missing the man.

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Developmental Psychology

ELIZABETH B. HURLOCK, PH.D. 1959, *second edition*. New York: McGraw-Hill Book Co. 630 pages. Illustrated. \$6.75.

The human life span from conception to old age is reviewed by developmental stages in this second edition of Hurlock's text. Instructors of college classes in psychology and human development will recognize much of the material. Familiar concepts and common sense observations are well supported by research studies, many of which are quite recent. On the other hand, some fallacious popular beliefs are dispelled by scientific evidence.

The characteristics and special problems of each developmental stage are described, with repeated warnings that the sequence of development but not the rate can be predicted. The author is careful to point out that there are individual differences among normal people at any age but that some common characteristics can be defined.

Physical, emotional, and intellectual facets of personality are explored. Interests, activities, needs, attitudes, and behavior patterns are discussed for each of the various stages and for the two sexes. Environmental influences are emphasized. The effect of early experiences upon adjustment in later years is stressed. Criteria are given for assessing successful adjustment. Possible reasons for deviant behavior are presented. The author asserts that well-adjusted and maladjusted people encounter about the same stresses, strains, and problems. The manner of coping with them is the distinguishing difference.

All ages, including adulthood, middle age, and old age, are said to bring their own particular adjustment problems. Fulfillment in middle years and old age is possible, says Hurlock, if the individual remains interested in learning new things and participating in new activities. Intelligence, she reports, does not decline with age, and judgment may even improve. Contrary to popular belief, many people are more liberal in middle age than they were in youth. Balancing their gradual decline in energy is their experience, social responsiveness, and dependability.

Problems of older people often spring from unfavorable attitudes toward aging. Such attitudes are greatly influenced by cultural, economic, and social conditions. For example, in a prosperous era, attitudes toward the aged tend to be respectful. Psychologic states can hasten or delay the aging process. Self-concept, social role, and motivation are among the important factors in a satisfying life in any stage of development, including old age.

The book is well organized. The similarity of outline for discussing each developmental stage makes it easy for the reader to locate topics of specific interest. Each chapter is followed by a rich bibliography. The style of the writer is straightforward, and the language is relatively simple. Understanding is enhanced by the liberal

use of charts, graphs, and illustrations. At times, the discussion becomes somewhat repetitious, but, in the main, this book will be a very useful classroom text and a valuable reference for professional people. It assembles and integrates a wealth of information which would otherwise be available only from scattered sources.

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Chronic Illness in a Rural Area

RAY E. TRUSSELL, M.D., and JACK ELINSON, PH.D., 1959. Cambridge, Mass.: Harvard University Press. 383 pages. \$7.50.

To learn the basic information on chronic illness in a rural community presented a distinct challenge. Drs. Ray Trussell and Jack Elinson have written, in a logical and concise manner, the objectives, the plans and their modifications, the surveys, and the analyses of a survey conducted in Hunterdon County, New Jersey. The authors' report demonstrated the teamwork of the Hunterdon Medical Center, the National Opinion Research Center, the New Jersey State Department of Health, the United States Public Health Service, and hundreds of local volunteers.

The objectives were twofold: to advance the methodology of morbidity surveys and to obtain new estimates of the prevalence of chronic illness and disability in a rural population, the medical and paramedical care needed by such a population, and the rehabilitation potential of the chronically ill and disabled.

The plan to attain the objectives included a community self-survey, family interviews, medical verification, clinical evaluations, statistics, and analyses.

Hunterdon County, with its population of 42,736, is cared for by 25 active general practitioners and has no local hospital, laboratory, diagnostic facilities, rehabilitation services, health department, or medical specialists.

The illustrations and charts demonstrated concretely the more-than-estimated number of people with chronic disease. The dramatic aspect, however, was found by appraisal of the preventability of diseases and the previous medical care received by the sample of persons with medically disabling conditions as determined by clinical evaluations.

This book is highly recommended to all physicians. To those in public health, it clearly shows the need for continued effort and performance in the ideals of public health and preventive medicine. To physicians in private practice, it illustrates their important position in therapeutic and preventive medicine. Last, but not least, to medical students, it enlarges the vista of that to come—the heritage, failures, and achievements attained and to be attained.

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Autogenous Vein Grafts

W. ANDREW DALE, M.D., 1959. Springfield, Ill.: Charles C Thomas. 117 pages. \$6.00.

This book discusses important aspects of peripheral vascular grafting with emphasis on the use of autogenous veins as arterial replacements. In addition to a discussion of the historic development of grafting technic, there are chapters on the pathophysiology of vascular insufficiency, arteriographic examination, operative technic, pre- and postoperative care, and, most important, the author's extensive personal experience with autogenous veins used as arterial grafts. The accumulated experiences of many surgeons using homografts and synthetic prostheses are compared to results obtained when autogenous veins are used to replace occluded arteries. In considering the various materials available at present for arterial grafting, it is concluded that, because of late degeneration and limited availability of homologous arteries, synthetic tubes are preferable as replacements. Autogenous veins, when used for this purpose, show little tendency to undergo degeneration. Adequate patency has been demonstrated if the vascular replacement involves only a short segment.

Further experience is necessary before conclusions can be reached for long segments of vein grafts. This monograph concisely and interestingly summarizes many important aspects of peripheral vascular disease and is recommended for individuals interested in this subject.

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Clinical Disorders of Hydration and Acid-Base Equilibrium

LOUIS G. WELT, M.D., 1959. second edition. Boston: Little, Brown & Co. 325 pages. \$7.00.

In this new epoch of science, dedication to a well-defined hypothesis is the key to success, for science advances, as armies do, by turning a flank and attacking the breach in force. The booty is a mixture of both relevant and incidental information, useful only when sifted and integrated. *Clinical Disorders of Hydration and Acid-Base Equilibrium* is a product of careful sifting of basic literature and clinical experience. The author does not dilly-dally, evade issues, or evoke dogma to avoid controversy. He assembles the major tenets of the past five years, interprets them, and reissues them systematically and succinctly.

Although the subject matter ranges from colligative properties of solutions to counter-current theory and treatment of edema and acute renal insufficiency, the various themes are so remarkably interwoven that there is no sensation of passing from one chapter to the next. The problem of water and electrolyte balance is not treated as an exercise in dialectics, nor is it dispensed halfheartedly with some such fixed aphorism as "when in doubt, give the patient a liter of isotonic saline by vein at the rate of 250 ml. per hour." The author knows that therapy cannot, except by chance, be rational without a trace of understanding. He properly discusses disturbances in hydration and acid-base balance in terms of effects on physicochemical forces and resultant compensatory mechanisms.

Introductory chapters serve to orient the reader. They deal with volume and composition of body fluids, exchange of water and electrolytes, and normal acid-base equilibrium. These are followed by a summary of modern concepts of renal physiology. The clinical chapters on

dehydration, edema, derangements in acid-base balance, and acute renal insufficiency which follow are thus rendered meaningful and are not merely "cookbook" instructions on how to be a practitioner à la Welt. A chapter on "Special Problems Presented by the Pediatric Patient," by Robert W. Winters, broadens the scope of the book and helps to make it a useful monograph for the medical practitioner.

EDWARD J. COFRUNY, P.H.D.
Ann Arbor

NEW BOOKS RECEIVED

Books and publications received will be listed here periodically. Those of special interest to our readers will be reviewed as space permits.

Baby Name Finder. J. E. SCHMIDT, M.D., 1960. Springfield, Ill.: Charles C Thomas. 390 pages. Illustrated.

Biochemistry of Human Genetics. G. E. W. WOLSTENHOLME, M.A., M.B., and CECILIA M. O'CONNER, B.Sc., Editors, 1960. Boston: Little, Brown & Co. 328 pages. Illustrated. \$9.50.

Cancer in Families. DOUGLAS P. MURPHY, M.D. and HELEN ABBEY, SC.D., 1959. Cambridge, Mass.: Harvard University Press. 76 pages. \$2.50.

Cancer of the Cervix. G. E. W. WOLSTENHOLME, M.A., M.B., and CECILIA M. O'CONNER, B.Sc., Editors, 1960. Boston: Little, Brown & Co. 109 pages. Illustrated. \$2.50.

Encyclopedia of Medical Syndromes. ROBERT H. DURIAM, M.D., 1960. New York: Paul B. Hoeber, Inc. 613 pages. \$13.50.

Enzymes in Health and Disease. DAVID M. GREENBERG, M.D., and HAROLD A. HARPER, M.D., Editors, 1960. Springfield, Ill.: Charles C Thomas. 459 pages. Illustrated.

From Fish to Philosopher. HOMER W. SMITH, 1959. Boston: Little, Brown & Co. 291 pages. Illustrated.

The Lifespan of Animals. G. E. W. WOLSTENHOLME, M.A., M.B., and CECILIA M. O'CONNER, Editors, 1960. Boston: Little, Brown & Co. 311 pages. Illustrated. \$9.50.

Modern Nutrition in Health and Disease. MICHAEL C. WOHL, M.D., and ROBERT S. GOODHART, M.D., 1960. Philadelphia: Lea & Febiger. 1084 pages. Illustrated. \$18.50.

The Low Sodium, Fat Controlled Cookbook. ALMA SMITH PAYNE and DOROTHY CALLAHAN, 1960. Boston: Little, Brown & Co. 429 pages. \$4.75.

Steric Course of Microbiological Reactions. G. E. W. WOLSTENHOLME, M.A., M.B., and CECILIA M. O'CONNER, B.Sc., Editors, 1959. Boston: Little, Brown & Co. 104 pages. Illustrated. \$2.50.

Virus Virulence and Pathogenicity. G. E. W. WOLSTENHOLME, M.A., M.B., and CECILIA M. O'CONNER, B.Sc., Editors, 1960. Boston: Little, Brown & Co. 105 pages. Illustrated. \$2.50.

The Journal Lancer

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NORTH DAKOTA, SOUTH DAKOTA, AND MONTANA

Series on COMMUNICABLE DISEASES

Mumps

LAURENCE G. PRAY, M.D.

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MUMPS is a common contagious disease, occurring most frequently during childhood, and is caused by a filtrable virus. It is mild in the majority of cases, characterized by swelling of one or both parotid or other salivary glands, low grade or moderate fever lasting for two or three days, some general malaise, and, sometimes, headache and nausea of a transient type. Fever may not be present at all in some cases and may rise to 102 or 103° in others. Acute symptoms usually subside within two or three days, and salivary gland swelling generally disappears within four or five days. There is almost always some local pain or aching which is marked in severe cases and in cases aggravated by obstruction of Stensen's or Wharton's ducts. A child sometimes has difficulty in completely opening his mouth and in chewing food. The throat may or may not be sore. Parotid involvement is bilateral in slightly over half of the cases; some authorities place this incidence as high as 70 to 80 per cent.^{1,2} In the majority of cases, the swelling occurs almost simultaneously in all affected salivary glands but, in occasional cases, secondary involvement may take place from two to seven days after onset. In approximately 10 per cent of cases, one or more submaxillary or sublingual glands may be involved instead of or in association with the parotitis.

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There are also instances in which orchitis, meningoencephalitis, or pancreatitis occurs without salivary gland involvement.

DIAGNOSIS

There is little or no difficulty in diagnosis of a typical case of mumps. Most cases in children are of the typical, full-blown variety, with parotid involvement, distinct ballooning of the cheek and jaw, and elevation of the ear lobe. This type can almost be diagnosed at a glance. The swelling is of a rather edematous but firm consistency without a discrete border, occurring in front of the ear as well as over the ramus of the jaw and below the ear. However, when parotid swelling is slight or only the submaxillary or sublingual glands are involved, diagnosis is more difficult. Submaxillary gland swelling is very easy to confuse with lymphadenitis in that region, but cervical lymphadenitis is characteristically more discrete and circumscribed than parotitis and involves the cervical rather than the parotid region.

Preauricular cellulitis or preauricular lymphadenitis may closely resemble mumps and cause errors in diagnosis. When parotid swelling is slight, one may confirm the diagnosis by palpating the ramus of the jaw, finding a rather doughy swelling covering the ordinarily sharp contour of the mandible. Examination of the opening of the salivary ducts is helpful, as there is usually redness and pouting of the opening of Stensen's or Wharton's ducts in cases of mumps.

Parotid gland tumors, Mikulicz's syndrome, pyogenic parotitis, and parotid stone may also have to be considered in atypical cases. One must also remember that a previous mumps infection does not invariably rule out the possibility of a recurrence, as there have been confirmed, though rare, cases of recurrent mumps in the same individual. Demonstration of an elevated serum amylase during the active course of mumps confirms the diagnosis, whereas a normal serum amylase rules it out. Complement fixation tests during the acute and convalescent stages of the disease establish the diagnosis of mumps if a rise of titer occurs during convalescence. Skin testing with mumps antigen during the acute and convalescent stages serves the same purpose.

CLINICAL FEATURES

The incubation period is exactly eighteen days in a majority of cases, with a range of seventeen to twenty-one days as the customary limits. One would tend to question either the diagnosis or the time of exposure if the incubation period were less than fourteen or greater than twenty-one days. There may or may not be a prodromal period of one or two days, with fever, generalized aching, nausea, and headache. In most cases, there is no such prodromal phase, and the child is first noticed to be somewhat listless and feverish at the time that swelling of the parotid gland is first evident. In some cases, there is no fever or any appreciable malaise.

Stokes¹ divides patients with mumps roughly into 5 groups: (1) those with a short course, whose signs and symptoms are insignificant; (2) those in whom the disease is full-blown, with salivary gland swelling but no complications; (3) those with severe mumps complicated by epididymo-orchitis, meningoencephalitis, or both; (4) those with no apparent symptoms but with typical responses of antibodies; and (5) those with meningoencephalitis or orchitis without involvement of the salivary glands.

The white blood cells are either normal or decreased in uncomplicated cases of mumps, with a relative lymphocytosis on the differential smear. In cases with acute complications, white blood cells are increased and polymorphonuclear cells are relatively more numerous.

COMPLICATIONS

Meningoencephalitis is the most frequent complication of mumps, estimated to occur in 30 to 40 per cent of cases if one includes patients with transient headache and fever as well as those with typical high fever, headache, stiff neck and

back, positive Kernig's signs, and vomiting. Meningoencephalitis may occur in the absence of salivary gland swelling and thus arouse suspicion of poliomyelitis or some other type of aseptic meningitis. Spinal fluid cell count may be normal or increased to more than 500 cells per cubic millimeter, with lymphocytes predominating. Spinal fluid pressure is moderately increased. Sugar and chloride levels are normal in the spinal fluid, but protein content is moderately elevated. Finkelstein² demonstrated pleocytosis in 16 of 40 patients with mumps, 6 of whom had no central nervous system symptoms. Others have confirmed this relatively frequent occurrence of meningoencephalitis in mumps. Fortunately, this complication is usually self-limited, subsiding without sequelae in five to seven days. However, there are rare instances of fatal central nervous system complications. Table 1, by Meade,⁴ includes these complications as well as other rare manifestations which should be borne in mind.

Orchitis or epididymo-orchitis almost never occurs in childhood but is a feared complication in postpubertal males, occurring in 18 to 20 per cent of cases, two-thirds of which are unilateral. It may begin before, during, or even several days after salivary gland swelling has subsided, and it sometimes even makes its appearance without any accompanying or preceding sialadenitis. It is manifested by severe local pain and swelling accompanied by sudden onset of chills and high fever. Symptoms remain acute for several days

TABLE 1
MANIFESTATIONS OF MUMPS

<i>Glandular or parenchymal</i>	<i>Nervous tissue</i>	<i>Other</i>
Parotitis	Meningitis	Myocarditis
Submandibulitis		Arthritis
Sublingual sialitis		Skin rashes
Orchitis		
Pancreatitis		
Oophoritis		
Prostatitis		
Epididymitis		
Seminal vesiculitis		
Hepatitis		
Thyroiditis		
Mastitis		

COMPLICATIONS OF MUMPS

Meningoencephalitis	Thrombocytopenic purpura
Peripheral neuritis	Sterility
Cranial neuritis	Urethral obstruction
Myelitis	Laryngeal edema

to a week. According to Mcade,⁴ atrophy of one testis occurs in nearly 10 per cent and sterility in less than 1 per cent of cases. Oophoritis in the postpubertal female is considerably less frequent than orchitis in the male, occurring in about 5 per cent of cases. Inasmuch as the ovary is not enclosed in a dense, fibrous sheath, such as the tunica albuginea, which encompasses the testes in the male, sterility is not a complication of oophoritis and acute symptoms are less severe.

Pancreatitis is a complication of mumps in 5 per cent of patients of all ages, although usually milder in children than in adults. Epigastric pain and tenderness, nausea, and vomiting are characteristic findings. High fever and up to 30,000 to 40,000 leukocytes per cu. mm. are present with severe involvement. Symptoms ordinarily subside in three or four days.

EPIDEMIOLOGY

The disease is both epidemic and sporadic, usually occurring in the late winter and spring months. It is not nearly as contagious as measles or chicken pox, close contact usually being required for spread of infection. Transmission of the virus is by direct contact, droplet infection, or, occasionally, by fomites, which gain entry by way of the nose, throat, or eyes. For that reason, epidemics are not as widespread as with some other contagious diseases, although the majority of children do contract mumps sometime during their preadolescent school years. Epidemics are said to occur every seven or eight years, primarily in the age group between 4 and 15 years. Many adults are immune to mumps even without a personal history of the disease because of the frequency of mild and unapparent infections. One attack usually gives lifelong immunity regardless of the number of salivary glands involved. As mentioned previously, there are substantiated rare exceptions to this rule.

The number of persons susceptible to mumps varies inversely to the density of population. The majority of military personnel contracting mumps are those from rural areas who were never exposed to the virus throughout their childhood. There is no sex difference in susceptibility. There is transplacental immunity in infants up to the sixth or seventh month, though one occasionally sees mumps occurring in the neonatal period if the mother herself is not immune or if she has the disease at the time of delivery.

THE VIRUS

The viral etiology of mumps was determined by the experiments of Johnson and Goodpasture in 1934.⁵ Enders⁶ later cultivated the virus on the

allantoic sac of the chick embryo. He was also able to transmit the disease to monkeys by inoculating the virus into Stensen's duct and by way of the nose and throat. In addition to growing in both the allantoic and amniotic sacs of the chick embryo, mumps virus may also be grown in cultures of Hela cells as well as some other human and simian tissues. The virus has been shown to be 90 to 135 m μ . in diameter and passes through Berkefeld V and N filters. It contains two antigens: a small soluble S antigen attached to chick membranes and a large V antigen attached to the virus itself. The antibodies to the S antigen appear first and disappear within six to twelve months from the time of the acute disease, while the antibodies to the V antigen appear later and decrease in four to six months to a lower level of 1 to 4 or 1 to 8, where they remain throughout life in most persons. The complement fixation test developed by Enders shows the rise of both antibodies in convalescence three to six weeks after the acute stage. A skin test for mumps was also developed by Enders from an antigen prepared from the parotid gland of previously infected monkeys and is now prepared commercially from infected perieembryonic fluid of chicken eggs. The skin test causes a positive reaction in the immune individual and a negative reaction in the susceptible person. A positive test appears in twenty-four to thirty-six hours and consists of an area of erythema, with or without induration, 1.5 cm. or more in diameter. Pseudoreactions may develop in those who are hypersensitive to egg protein.

PREVENTION

In view of the greater danger of complications of mumps in the adult as compared to mumps in the child, it is advisable to try to expose all prepubertal children to the disease.^{1,7} If an adult without a previous history of mumps has close contact with a case in the home or elsewhere, an attempt should be made to determine susceptibility and to immunize if susceptible. Immunization with formalin inactivated mumps vaccine early in the incubation period has a chance of giving appreciable immunity before mumps develop. Two injections of 1 cc. each are given subcutaneously five to ten days apart. My own experience with this method is limited to one case, in which a susceptible father was exposed to his young child with mumps. He was given the vaccine as described. The first dose being administered one or two days after exposure. A mild parotitis developed on the eighteenth day, but he had no fever or generalized symptoms

and orchitis did not develop. Ideally, immunization should be carried out prior to exposure and a booster shot given when exposure occurs.

Convalescent serum or gamma globulin is not of proved value in prophylaxis, but it may be helpful. Ordinary pooled gamma globulin is ineffective.

The patient is considered contagious as long as there is any salivary gland swelling or fever. The disease may also be transmitted for twenty-four to forty-eight hours prior to the development of salivary gland swelling. Isolation of postpubertal children and adults is recommended for a minimum of one week, providing the symptoms and swelling have disappeared by that time. Children usually return to school in a week to ten days, depending on the severity and duration of their symptoms.

TREATMENT

Bed rest is advised during the period of salivary gland swelling and fever. Hot or cold applications to the swollen area usually give some relief from aching or pain, the latter being generally more effective. Aspirin is helpful in relieving discomfort; in severe cases, codeine or its equivalent is used to reinforce the effect of aspirin. Sedatives are occasionally required at night. Liquid and soft foods usually are well tolerated.

Meningoencephalitis is treated symptomatically. Spinal puncture, with removal of several cc.'s of cerebrospinal fluid, often relieves the severe headache. To relieve pain in cases with orchitis, the swollen testicle is suspended in a cradle of tape attached to the thighs or rests on a nest of cotton between the thighs; an ice bag is also used. Analgesics and sedatives are prescribed, and intravenous fluids are given when vomiting causes dehydration. There is general agreement that incision of the tunica albuginea helps to relieve pain and prevents pressure necrosis of the testicle. There is no evidence at present that adrenocortical steroids, ACTH, or antibiotics are

of any value in either treatment or prevention of mumps or mumps orchitis.

Pancreatitis is treated symptomatically with intravenous fluids, usually indicated because of vomiting. Pain and discomfort are treated with analgesics and sedatives. Other complications are rarer and treated as indicated by localization and severity. Fatalities are rare but have been known to occur in complicated cases. Complete recovery is the rule.

SUMMARY

Mumps is an acute contagious disease which is usually mild in childhood but is often accompanied by severe complications in the adult. It is a viral disease, the virus being grown in chick embryos and in monkeys.

Complement fixation tests and mumps skin tests are of value in establishing the diagnosis of mumps. An elevated serum amylase is reliable confirmatory evidence of the disease, whereas a normal serum amylase helps rule out the diagnosis.

Mumps is to be avoided in adults, particularly in males. For this reason, deliberate exposure of preadolescent children is advised. Active immunization of adults is now possible, preferably prior to exposure. Treatment of mumps is largely symptomatic, although complications sometimes need specific therapy for relief.

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THE DIAGNOSIS of primary miliary tuberculosis may be established by liver biopsy before respiratory symptoms occur. Vim-Silverman needle biopsy of the liver yielded numerous granulomas suggestive of tuberculosis in one patient with negative sputum and chest roentgenograms. Acid-fast bacilli were found on culture of a second biopsy specimen. About six weeks later, a chest roentgenogram revealed upper lobe infiltration and sputum became positive.

R. J. HEALY, A. H. LEFF, and B. D. ROSENAK: Needle biopsy in tuberculosis of the liver, with culture of acid-fast bacilli. *Am. J. Digest Dis.* 4:638-641, 1959.

The Hemiplegic Patient

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ASPASTIC WEAKNESS of one side of the body is one of the most common neurologic problems encountered in the practice of medicine. It is due to a lesion within or adjacent to the motor system contralateral to the side of involvement. The patient may offer a wide variety of complaints, mostly related to the decreased facility in using the involved limbs because of increased tone. In slowly progressive lesions, the involvement may be very severe before the patient actually seeks help.

Examination usually reveals very little motor weakness. Functional use of the involved limbs will be decreased because of the spasticity. The tone of the involved muscles will be increased and automatic movements, such as swinging the limb, will be reduced. The entire limb will be involved. The deep reflexes are hyperactive, and the toe signs are generally positive on the involved side. The actual clinical diagnosis of hemiplegia or hemiparesis is not difficult, but the etiologic process causing this motor system deficit may be a problem, particularly since therapy must be directed toward the specific etiologic process. Actually, the best guide to etiology is the age of the patient, since different pathologic processes are characteristic of different age groups.

CLASSIFICATION OF HEMIPLEGIA

Infancy, birth to 2 years

1. Birth injury (cerebral palsy)
2. Subdural hematoma

Childhood, 3 to 10 years

1. Emboli (rheumatic heart)
2. Brain abscess

Adolescence, 11 to 20 years

1. Trauma

Youth, 21 to 35 years

1. Multiple sclerosis
2. Tumors

Middle age, 36 to 65 years

1. Multiple sclerosis
2. Thrombosis of internal carotid artery
3. Subdural hematoma
4. Tumors
5. Vascular disease (hypertension)
6. Syphilis

Old age, 66 years and over

1. Vascular disease (thrombosis, hemorrhage)
2. Tumor
3. Subdural hematoma
4. Syphilis
5. Thrombosis of internal carotid artery

INFANCY

Cerebral palsy. This is not a disease but a symptom complex due to brain injury at or before birth. These children may present a wide variety of neurologic symptoms, but almost half will show some spasticity, often unilateral.

This disorder, if mild, may not be apparent at birth. It should be suspected in any infant with a history of difficult birth with cyanosis, convulsions, feeble cry, or poor nursing. The diagnosis is established by the objective evidence of impairment of motor function and generally can be made at 6 months to 1 year of age. The average normal child grasps objects at 3 months and reaches for objects at 5 to 6 months. He stands at 14 months and walks at 15 months. It is in carrying out these activities that spasticity and paucity of movement will be detected on the involved side. The nature of the involvement generally can be determined in the latter half of the first year by the muscular hypertonicity, hyperreflexia, and clonus.

In mild cases, the diagnosis may not be apparent for years, although the parents may have noticed decreased unilateral motor activity for a long time. In many children, unilateral hemiparesis is accompanied by ataxia, athetosis, or retarded intellectual development indicative of more widespread cerebral damage.

Usually, this condition appears to be progressive during the early years of life and then be-

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comes stationary or improves as the child develops compensatory mechanisms to help overcome the physical disability.

Subdural hematoma. This is a commonly overlooked syndrome, chiefly because the history of precipitating trauma is often not obtained. The symptoms may be slow or sudden in onset and consist of restlessness, enlargement of the head, convulsions, motor weakness, and tense fontanelles. Any infant who shows *no signs of a systemic illness* and develops *motor weakness* should be suspected of having a subdural hematoma.

Diagnosis is readily established by subdural taps through the fontanel. Symptoms are relieved by evacuating and removing the hematoma.

CHILDHOOD

Emboli. These lesions are caused by an infectious valvulitis. The original systemic disease results in a number of suggestive symptoms and signs, such as painful joints, weight loss, irregular fever, leukocytosis, accelerated sedimentation rate, petechiae, and cardiac murmur. In such a patient, the acute onset of convulsions, lethargy, or hemiplegia should promptly suggest a diagnosis of cerebral emboli. Diagnosis can be established by blood culture.

Treatment should be directed toward the primary illness and consists of large doses of antibiotics. As the primary illness is brought under control, the cerebral symptomatology also improves.

Brain abscess. This diagnosis should always be suspected in a child with *chronic middle ear, mastoid*, heart, or lung involvement who suddenly develops convulsions, *hemiplegia*, and/or *signs of increased intracranial pressure*. Diagnosis is usually confirmed by the signs of sepsis, such as chills, fever, leukocytosis, and mental confusion. The finding of increased cells in the spinal fluid, particularly leukocytes, completes the diagnosis.

Treatment should consist of surgical evacuation of the abscess. Operation is often delayed until the abscess is partly encapsulated, and, in the interval, the patient is treated with large doses of antibiotics.

ADOLESCENCE

Trauma. Hemiplegia or hemiparesis occurs in about 10 per cent of all head injuries. In civilian life, this age group is involved chiefly as a result of injuries secondary to sports and, less commonly, to automobile accidents. In most cases, diagnosis is apparent by the history of the injury. When no history of injury is obtained,

trauma must still be kept in mind and the patient carefully checked for *bruises, stiff neck, skull fractures, or blood-tinged spinal fluid*. A combination of any of these conditions should strongly point to trauma as the cause of the motor weakness.

The motor weakness is often accompanied by headache, mental confusion, and vertigo. These symptoms disappear after a few weeks. The motor weakness also improves slowly, unless the brain damage is severe.

Treatment is symptomatic. The patient should be activated as soon as his condition permits. Exercises should be given for the impaired musculature and continued after hospital discharge.

YOUTH

Multiple sclerosis. This degenerative disease is one of the most common causes of acute motor weakness in this age group. A careful history usually reveals the previous presence of *other transient neurologic complaints*, such as vertigo, visual disturbances, paresthesia, and so forth. As a rule, the neurologic examination discloses evidence of *scattered involvement* of the nervous system, such as bilateral positive toe signs, reflex irregularities, and nystagmus. The presence of recurrent scattered disturbance of the nervous system in a young patient with motor weakness characterizes this disease.

Tumors. Although not common in this age group, a brain tumor must be considered, particularly in the presence of a *slowly progressive* development of *motor weakness* as an isolated finding. The two tumor types that most frequently occur in this age group are the astrocytoma and the meningioma. Diagnosis should be suspected from the history and verified by special laboratory studies, such as air studies or angiography.

MIDDLE AGE

Multiple sclerosis. This demyelinating disease may also produce an acute hemiplegia in patients in the fourth decade of life. However, the diagnosis should be made with great caution if this is the first episode of neurologic involvement. Invariably in multiple sclerosis, the patient has had previous episodes of neurologic disturbances and an examination will show evidence of a more widespread neurologic disease.

Thrombosis of the internal carotid artery. Since each cerebral hemisphere receives a blood supply from both internal carotid arteries through the circle of Willis, the symptoms resulting from occlusion of a single internal carotid artery will

depend upon the efficiency of the collateral circulation. In individuals of this age group, the collateral circulation is generally good, so that occlusion of this artery usually results in only a *mild transient motor weakness*.

This syndrome may appear in three distinct patterns:

1. "Transient or sputtering" form, in which there are recurrent episodes of motor weakness lasting hours to days. Such a series of attacks may recur over a period of months or years but ultimately will terminate in a permanent hemiparesis if not adequately treated.

2. "Apoplectic" type, in which there is a sudden, permanent motor weakness associated with headache and even disturbances of consciousness. Usually improvement ensues in a few weeks.

3. "Progressive" form, which presents as a slowly progressive motor weakness over a period of weeks and resembles the picture produced by a brain tumor.

The "sputtering" type is easily diagnosed. The other two types may be difficult to diagnose. It can be aided by careful palpation of the internal carotid artery through either the neck or the inside of the throat. Absolute diagnosis can be made by means of angiography.

The transient type is treated with an antieagulant or by surgical removal of the occluding thrombus. Once the occlusion is complete, treatment is symptomatic. The prognosis is usually good, and most patients recover considerable motor function.

Subdural hematoma. This condition should be considered in any patient in this age group with a *history of trauma or alcoholism* who develops signs of progressive motor weakness. In addition to the motor symptoms, headache, forgetfulness, and clouding of the sensorium develop. The diagnosis can be established by the appearance of a shift of the pineal gland on the skull roentgenogram or by air studies or angiography.

Treatment consists of evacuation of the lesion.

Tumors. In this age group, brain tumors comprise an important and relatively frequent cause of motor weakness. The most frequent tumors are either rapidly growing gliomas or metastatic lesions, which carry a poor prognosis. The symptoms usually appear acutely with *rapid development of motor weakness* over a period of weeks or a few months and are often accompanied by headache, lethargy, and increased intracranial pressure.

The diagnosis can be substantiated by (1) careful history or examination for a primary lesion, (2) roentgenogram of the chest for me-

tastases, (3) roentgenogram of the skull for a pineal shift, and (4) angiography. In a metastatic lesion, the most frequent sources of the primary lesion are the lung, breast, gastrointestinal tract, and skin, and these areas should be carefully studied for a primary lesion.

Vascular disease. Cerebrovascular disease is a relatively uncommon cause of motor weakness before the age of 65 years. In patients with severe hypertension, intracerebral hemorrhage may occur in this age group and produce dramatic symptoms, in which hemiplegia plays an important role.

These patients often show mild prodromal symptoms of postural vertigo and headache. The hemiplegia occurs suddenly, often during activity, and is accompanied by intense headache and impaired or complete loss of consciousness. The neck is mildly stiff, and the spinal fluid may be blood-tinged and under increased pressure. The deep reflexes on the involved side may be entirely absent and the paralyzed limbs flaccid. If the patient survives this episode, the paralyzed limbs soon become spastic and the deep reflexes hyperactive.

The over-all prognosis with an intracerebral hemorrhage is very poor. Over 75 per cent of the patients die within the first week. If the patient survives, the prognosis for functional improvement is very good.

Syphilis. At the present time, this disease is an infrequent cause of motor weakness but must be considered in any complete differential diagnosis of this symptom complex. Both general paresis and meningovascular syphilis can produce hemiparesis. The diagnosis should strongly be considered in any patient with motor weakness who (1) has a history of having had syphilis or having been treated for the disease, (2) shows papillary irregularities, or (3) has a positive blood or spinal fluid serology.

Both forms of this disease respond well to penicillin in doses of 15 to 20 million units administered over a three-week period. The motor weakness in each form usually clears up promptly.

OLD AGE

Vascular disease. Cerebrovascular accidents, chiefly cerebral thrombosis, predominate as a cause of hemiplegia in this age group. Often for years before onset of the cerebrovascular accident, the patient has prodromal symptoms, such as headache, postural vertigo, and poor memory. The hemiplegia usually develops slowly while the patient is at rest. There is no loss of consciousness and no evidence of increased intracranial pressure. Most of these patients survive,

and about 85 per cent can be rehabilitated so that they can be cared for in their normal environment.

Tumors, subdural hematoma, and syphilis also occur in this age group. Diagnosis and prognosis are the same as already discussed in the previous age group.

TREATMENT OF THE HEMIPLEGIC PATIENT

Hemiplegia, once it occurs, may persist even though the etiologic factors have been removed. Hence a patient cured of his illness, such as by removal of a hematoma, may still have persistent hemiplegia that requires additional attention and therapy. It is to be kept in mind that no patient with hemiplegia should remain bedridden. Every hemiplegic patient, with a little guidance from the physician, can be taught to ambulate and to care for himself.

The immediate care of the hemiplegic patient consists of: (1) a diagnosis as to etiology and treatment aimed at the causative factors, (2) general medical care, (3) good nursing care, and (4) rehabilitation.

Diagnostic and therapeutic procedures have already been discussed. Medical therapy for the underlying disease is begun immediately upon arrival at the hospital. Nursing care is of utmost importance in order to: (1) prevent contractures (by passive movement of extremities through a full range of movement initiated immediately on admission to the hospital); (2) avoid hypostatic pneumonia (by frequent change of the patient's positions, removal of nasal and oral secretions, and administration of antibiotics); and (3) prevent decubital ulcers (by close attention to skin hygiene). The nursing staff can also aid recovery by instituting a schedule for eating, bowel habits, waking, and sleeping as the patient begins to improve.

Most hemiplegic patients, depending upon the severity and cause of the involvement, are kept in bed from three to six weeks. As soon as the acute symptoms have subsided and while the patient is still bedridden, passive movement should be instituted to prevent deformities. Between therapy periods, footboards, sandbags, and splints may be used to maintain proper position of the limb. As the patient improves, he is encouraged to use the affected extremity as much as possible and to care for his bedside needs. Heat and massage may be used for painful extremities.

Ambulation is started when the patient is strong enough to bear weight on the affected

limb. He usually is started by balancing on the affected limb either with support or in parallel bars and slowly progresses through the stage of ambulation with walking aids, such as crutches or a walker, to walking without aids.

The process of rehabilitating the hemiplegic patient is an important part of the total treatment program and must be instituted in each case. It generally can be carried out even in the home, provided the physician understands his goal, is sympathetic, and is willing to spend a little time with the family and the patient in outlining and directing the course of therapy. Members of the family can be taught to carry out passive movement in appropriate cases. Some patients, particularly those with milder cases, may not need a trained therapist to teach them self-care and ambulatory activities and may be assisted instead by the family under the supervision of the physician.

The outlook for functional recovery naturally varies from patient to patient and can be determined only by careful neurologic evaluation from time to time. All patients with hemiplegia, however, regardless of the degree of recovery, are capable of ambulation if given adequate rehabilitation therapy.

There are a few complications that may occur and should be kept in mind in evaluating the final prognosis in any single case. *Thalamic pain* is a very unfortunate complication and is due to involvement of the thalamus by a deep-seated lesion. This complication occurs about four to six months after hemiplegia appears and may be intractable to therapy. It consists of a burning, searing pain that most often involves the upper extremity and face but may implicate the entire half of the body. This type of complication should be considered in any patient with hemiplegia associated with hemihypesthesia suggesting a deep-seated lesion in the thalamic region. *Seizures* are an uncommon complication of hemiplegia; when they occur, they generally can be controlled by anticonvulsants. In right-sided hemiplegia, *speech disturbances* frequently occur. This speech involvement often accentuates the degree of incapacitation of the patient and complicates rehabilitation. Speech therapy is often helpful and should be instituted early. *Intellectual impairment* is by far the most serious complication. It usually occurs in individuals who have had diffuse cerebral damage prior to onset of hemiplegia. The prognosis in such patients is poor, and rehabilitation usually is not successful.

Common Cerebrovascular Syndromes

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CEREBROVASCULAR DISORDERS comprise one of the most common groups of illnesses encountered in general practice. Cerebrovascular disease may be due to a large number of causes, each of which must be considered in the evaluation of every case. It might be well to briefly consider some of the general causes of cerebrovascular disease before discussing the individual entities.

GENERAL ETIOLOGY OF CEREBROVASCULAR DISEASE

Disorders of the heart. Any disease process that diminishes cardiac output may also reduce cerebral blood flow and result in transient or permanent cerebral symptoms. This type of mechanism is active in such conditions as Stokes-Adams syndrome, orthostatic hypotension, aortic stenosis, or cardiac arrest.

Disorders of the blood vessels. A large number of different disorders of the blood vessels themselves can result in cerebral symptomatology. By far the most frequent is *narrowing* or *occlusion* as is seen in such conditions as arteriosclerosis, syphilis, and some of the rarer collagen diseases, such as thromboangiitis obliterans, periarteritis nodosa, and lupus erythematosus. Cerebral thrombosis secondary to atherosclerosis is by far the most frequent of these conditions.

Emboli. Different types of emboli may also interfere with cerebral circulation and produce symptoms. By far the most frequent type is caused by cardiac vegetations, either valvular or mural. However, many types of emboli have been reported to result in cerebrovascular occlusion, such as fat, tumor, and air emboli and even parasites, such as occur in malaria.

Alterations in the vessel wall. These changes may cause cerebrovascular disease. This is particularly true in the cases of aneurysms which often rupture, resulting in dramatic cerebral symptomatology. Toxic processes, such as heavy

metal and industrial poisons, may so alter the vessel wall as to result in complete vascular thrombosis or hemorrhage.

Changes in the blood properties. Any condition that causes a change in the blood properties may result in cerebrovascular disease. In polycythemia, for example, the increased viscosity often produces cerebral thrombosis. The same condition is seen in severe cases of dehydration, particularly in older individuals. In cases of intracranial hemorrhage, certain drugs, such as the anticoagulants, occasionally produce a tendency toward bleeding. A similar bleeding tendency is observed in certain diseases, such as hemophilia and purpura.

SPECIFIC VASCULAR SYNDROMES

Infancy up to 2 years

Subdural hematoma. This is the only consistent vascular syndrome encountered in this age group. This condition usually follows trauma but is also associated with debilitating diseases, dysentery, pneumonia, anemia, and cachectic states. In many infants, no etiologic factors can be elicited, and the diagnosis must be suspected from the clinical picture.

Clinically, these infants show no evidence of any systemic involvement. At the onset, they are afebrile but suddenly become restless, irritable, and feed poorly. Occasionally, the syndrome may present with vomiting or convulsions. Soon the fontanels begin to bulge, the sutures become separated, and the head slowly enlarges. In some cases, the infant may show evidence of focal cerebral involvement chiefly in the form of a slowly developing motor weakness. The diagnosis should always be suspected in any infant in whom evidence of cerebral involvement suddenly develops in the absence of a febrile illness. The diagnosis can be verified by subdural taps. Placing a needle into the subdural space through the fontanel often reveals bloody fluid under increased pressure.

Treatment consists of repeated aspirations of

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the subdural spaces until the bloody fluid is completely removed. It is then advisable to surgically remove the hematoma and its membrane. The prognosis depends upon the duration of the illness and the length of time taken to reach the proper diagnosis. The sooner the hematoma is removed, the better the outlook. Impairment of brain development with chronic residuals can be anticipated when removal of the hematoma has been delayed.

Childhood, 2 to 5 years

Sinus thrombosis. Primary or nonseptic thrombosis of the dural sinuses chiefly involves the superior longitudinal and straight sinuses. It may complicate a debilitating illness or may occur suddenly without specific cause. The onset is abrupt with a sudden loss of consciousness and convulsions, the latter being bilateral or unilateral. Signs of increased intracranial pressure with vomiting and papilledema soon develop. Edema of the forehead and distention of the veins of the scalp develop in some patients. Spinal puncture often aids in the diagnosis by revealing blood tinged fluid under increased pressure. Spasticity of both lower extremities may develop in some cases.

The severity of the neurologic symptoms as well as the prognosis depends upon the degree of involvement of the sinuses or the associated veins. In some cases, the motor defect is mild, resulting in paresis of a single limb and little or no increased intracranial pressure. In these patients, the prognosis is good and recovery may be complete with only mild residual weakness. The electroencephalogram may be helpful by indicating the extent of the cortical damage.

When the sinuses are extensively involved, motor symptoms may be bilateral, severe, and associated with definite increased intracranial pressure. Angiography may aid in the diagnosis by revealing a delay in the venous phase of the angiogram. Such cases carry a grave prognosis and many even prove fatal. Patients who recover may suffer residual recurrent seizures.

Treatment should be aimed at reducing the intracranial pressure. Recently, the use of a 30 per cent solution of intravenous urea has proved helpful. The urea is given in a dose of 1 gm. per kilogram of body weight by slow drip over a twenty-four-hour period.

Acute infantile hemiplegia. This is not a rare condition. It has been described in the literature under a variety of terms, such as Strimpe's disease, poliocencephalitis, and so forth. It is probably due to an arteritis of one of the branches of the middle cerebral artery, which

results in an inflammatory thrombosis and subsequent motor involvement.

This condition implicates both sexes equally and often appears in children who have been healthy prior to the illness. A seizure suddenly develops with profound loss of consciousness and an elevated temperature. The convulsions may recur, and the unconsciousness persists for days. The temperature usually subsides as the convulsions stop. The child slowly recovers but has a residual fairly complete hemiplegia often associated with hemianopsia and even speech disturbances if the lesion is on the dominant hemisphere. Laboratory studies are not helpful, and the spinal fluid as a rule is negative.

The hemiplegia usually persists as a permanent residual. Although most of these children do learn to walk and to use the involved upper extremity, definite limitations persist. The involved limbs often remain smaller and are occasionally disfigured. Rarely, there may also be some associated complications, such as speech disturbances, athetosis, or persistent convulsions. The intellectual development is usually not impaired, and most of these children adjust fairly well to their disability.

Treatment is symptomatic. A well-planned program of physical training and rehabilitation is necessary to obtain the maximum use of the involved limbs.

Youth, 6 to 15 years

Emboli. By far the most common vascular syndrome encountered in this age group is an embolus from valvulitis. It usually develops on the basis of a previous rheumatic endocarditis, such as a congenital malformation of the heart and may follow acute infections or surgical procedures upon the throat or teeth.

In many cases, the onset is insidious; there may be a long history of slight fever, malaise, weight loss, and joint pain. Occasionally, the valvulitis may be silent, and the first indication of the illness is the abrupt onset of the cerebral symptoms secondary to the emboli. The onset of the cerebral involvement is invariably abrupt with immediate lethargy or loss of consciousness often associated with focal involvement, such as motor weakness or hyperreflexia. Neurologic examination usually reveals evidence of diffuse cerebral damage with bilateral reflex changes and bilateral positive toe signs. The general examination offers confirmatory findings, such as leukocytosis, increased sedimentation rate, petechiae, and a cardiac murmur. The spinal fluid may contain an increase of both red cells and leukocytes.

Treatment with adequate doses of penicillin may cure many of these patients. The specific organism should be isolated and its sensitivity to the various antibacterial agents determined. Usually, very large doses of penicillin must be used, often 1,000,000 units every hour for a month. In some cases, even more prolonged therapy is indicated.

Young adults, 17 to 35 years

Subarachnoid hemorrhage. The most common vascular disorder of the young adult is subarachnoid hemorrhage secondary to a ruptured aneurysm. These small saccular aneurysms are produced by congenital defects of the vessel wall probably as a result of incomplete involution of the embryonic capillary plexus. Eighty-five per cent of these lesions arise from vessels of the anterior portion of the circle of Willis, such as the first bifurcation of the middle cerebral artery, the anterior communicating artery, and the origins of the posterior communicating, the middle, and the anterior cerebral arteries from the internal carotid stem.

These aneurysms usually remain silent until they rupture. They then produce a pathognomonic picture, which is initiated by an excruciating suboccipital pain associated with vertigo and nausea. The patient becomes restless, confused, or comatose. The neck is rigid, the Kernig's sign is positive, the pulse is often slow, and hyaloid or sheet hemorrhages may be present in the fundi. Shortly after the onset of the illness, the temperature may become elevated. The diagnosis is readily confirmed by spinal puncture, which reveals a bloody spinal fluid.

The typical acute rupture is readily recognized by the acute onset of occipital pain, confusion, and the associated stiff neck. However, in many cases, the bleeding is slow due to an incomplete rupture of the aneurysm. In such cases, the diagnosis may be more difficult but should be considered in patients of this age group who present: (1) recurrent frontal headache, often retro-orbital in location; (2) recurrent episodes of confusion associated with mild nuchal rigidity; and (3) acute frontal headache followed by diffuse pain in the lumbar and gluteal regions. In each of the foregoing situations, the diagnosis can be substantiated by the associated bloody spinal fluid.

The prognosis in ruptured aneurysm is grave; the average mortality rate in the initial attack is 40 per cent. Of the surviving cases, recurrent attacks occur in 50 per cent. The greatest frequency of recurrence is in the third week.

The immediate treatment consists of strict

bed rest, sedation, and other supportive measures until the patient's condition improves to the extent that further studies can be carried out. Carotid or vertebral angiography should be done to locate the aneurysm and to study the collateral circulation. Surgery can be considered if the location and size of the aneurysm is favorable.

Adults, 35 to 65 years

Insufficiency of the internal carotid or middle cerebral circulation. Isolated atherosclerotic plaques do occur on the large basilar vessels, producing a marked localized reduction of the vessel lumen. In the younger age groups, this isolated vessel narrowing tends to implicate primarily the internal carotid and middle cerebral arteries. In the presence of such a narrowing, a transient fall in blood pressure from any cause may reduce the blood flow through the narrowed vessel to such an extent that a part of the brain is deprived of its adequate metabolic needs. In such cases, symptoms of a transient insufficiency may occur even though the vessel is not completely occluded. If the internal carotid artery is involved, symptoms of insufficiency will result only if the collateral circulation is inadequate. This is seen chiefly in older individuals in whom the circulation through the circle of Willis has been reduced. The middle cerebral artery is an end artery and has no collateral circulation; therefore, a narrowing of this vessel, regardless of age will produce symptoms of insufficiency. Clinically, this type of vascular insufficiency results in a characteristic picture, consisting of recurrent symptoms which duplicate each other and persist from a few minutes to hours or days. The most common complaint is a transient unilateral muscular weakness or sensory disturbance, which, in subsequent attacks, implicates different extremities of the same side or the entire side. Speech disturbances chiefly manifested as an expressive aphasia may occur when the dominant hemisphere is involved. The speech involvement may antedate or follow the motor disturbance. Some patients may complain of episodes of transient blindness early in the disease.

Such a series of episodes may recur over a period of months or even years but ultimately terminate in permanent motor or sensory disturbances or even aphasia unless specific therapy is instituted before permanent occlusion results. The diagnosis should not be difficult, since the history and findings are pathognomonic.

Treatment must be instituted promptly and consists of the use of anticoagulants. The pro-

thrombin level should be kept at 15 to 25 per cent of the normal level by use of 50 to 100 mg. of Dicumarol daily.

Thrombosis of the internal carotid artery. Since each of the cerebral hemispheres receives blood from both internal carotid arteries through the circle of Willis, the symptoms resulting from occlusion of a single internal carotid artery will depend upon the efficiency of this collateral circulation and the condition of the opposite internal carotid artery. Younger individuals with good collateral circulation show few or no symptoms, while older individuals often have a severe functional deficit.

The most common complaint is the acute onset of some degree of unilateral muscular weakness which may first involve the upper extremity, although often the entire side may be implicated. Unilateral paresthesia or even hemihypesthesia may occur. An expressive aphasia may be present if the left internal carotid artery is implicated. Shortly after the onset of symptoms, a few individuals lose vision in one eye with primary atrophy of the optic nerve. Occasionally, mental symptoms predominate with little or no motor or speech disturbances. These patients manifest many vague complaints and present a picture of anxiety, depression, or even progressive deterioration with memory impairment, loss of drive, and personality changes. These symptoms may increase over a period of months, and only the associated occurrence of focal symptoms suggests the possible diagnosis.

The diagnosis of the apoplectic or the progressive forms of this disease may be very difficult but should be suspected in individuals of this age group. The diagnosis can be aided by palpation of the internal carotid artery either through the neck or the inside of the throat. This procedure may reveal a diminution or absence of carotid pulsation. Although the lack of such pulsation is diagnostic of this illness, the presence of good pulsations does not eliminate the diagnosis. The pull of the external carotid often permits good pulsations to be felt even when the internal carotid is thrombosed. Auscultation along the internal carotid artery in the neck may reveal a bruit at the site of the occlusion. Absolute diagnosis can be made by angiography, but this procedure may be dangerous in cases of partial occlusion.

Once thrombosis is complete, treatment must be symptomatic and is similar to that carried out for any stroke syndrome. In an acute thrombosis, surgical removal of the thrombus is often helpful. In the more chronic cases, good results can often be obtained by means of simple re-

habilitation procedures. Since most individuals do have some collateral circulation through the circle of Willis, most patients improve considerably and are able to walk and carry out most self-care activities.

Posterior inferior cerebellar artery thrombosis. This vessel is a branch of the cerebral artery and supplies blood to the lateral surface of the medulla. Because of the frequent and early atherosclerotic changes occurring in the vertebral artery, the blood supply to this vessel is often jeopardized with a temporary ischemia to those structures situated in this region of the medulla: namely, the eighth and tenth cranial nerve nuclei, the ipsilateral sensory fibers to the face, the ipsilateral cerebellar system, and the contralateral sensory fibers to the body.

These patients may complain of mild recurrent episodes of vertigo and paresthesia of the ipsilateral face for a few days prior to the acute illness. They then experience a sudden, severe vertigo that may cause them to fall to the ground. There is no accompanying loss of consciousness. Immediately following the acute attack, the patient has difficulty in swallowing and his face becomes numb. Examination discloses a loss of sensation over the face, a small pupil (Horner's syndrome), palatal weakness, and incoordination, all of which occur on the same side of the body. One can often elicit a loss of sensation over the limbs and trunk of the opposite side of the body with some contralateral hyperreflexia.

In many cases, only an incomplete syndrome may occur introduced by severe vertigo but revealing only a few of the foregoing findings, chiefly the facial sensory disturbance and the incoordination.

The prognosis is generally good. Invariably, the symptoms slowly clear up over a period of months, with incoordination often being the last disturbance to disappear. Occasionally, a retrograde thrombosis into the vertebral artery may result in grave complications. This is avoided by placing all of these patients on anticoagulant therapy for two to three months during the period of recovery.

Embolism from a mural thrombus. In this age group, most cerebral emboli are caused by mural thrombi resulting from an auricular fibrillation or a myocardial infarction. Less commonly, they may be due to atheromatous particles which break loose from the aorta or the great vessels of the neck.

Embolism is not often preceded by symptoms of cerebral circulatory disturbances, and prodromal symptoms are often lacking. The activity of the individual appears to play no role in the

process, except that emboli are occasionally dislodged from the fibrillating auricle at the time normal rhythm is restored.

The onset of symptoms is usually sudden and dramatic with loss of consciousness associated with scattered neurologic findings indicative of a bilateral and diffuse cerebral process. The deep reflexes may be unequal, and the toe signs may be bilaterally positive. Occasionally, the deep branches of the middle cerebral artery become occluded, resulting in hemiparesis or even transient aphasia. The spinal fluid generally is negative but, on occasion, may contain red cells. The diagnosis may be difficult and should always be suspected in this age group in the presence of a vascular syndrome producing diffuse cerebral involvement.

The prognosis in this type of cerebral embolism is generally good both for survival and recovery from the cerebral deficit. Treatment consists of anticoagulant therapy.

Age group over 65 years

Cerebral hemorrhage. This type of hemorrhage may occur spontaneously but often follows situations causing an increase in blood pressure, such as straining at the stool or lifting heavy objects; hence it often appears during the day while the patient is active.

With massive intracerebral hemorrhage, consciousness is usually rapidly lost due to the increase in intracranial pressure and destruction of brain tissue. In less severe cases, consciousness is lost more slowly, and intense headache may first develop associated with lethargy which deepens into a coma over a period of hours or even days. When the illness is fully developed, there is often evidence of brain stem involvement, such as slow pulse, and Cheyne-Stokes respiration. The spinal fluid is under increased pressure and is often blood-tinged. If the hemorrhage has ruptured into the ventricle or subarachnoid space, the neck may be rigid and the spinal fluid may be bloody. This is usually a bad prognostic sign.

The focal signs depend upon the site of the hemorrhage. The most common site is in the basal ganglia and internal capsule from rupture of one of the lenticulostriate arteries, which results in contralateral hemiplegia. During and immediately following the hemorrhage, all deep reflexes may be absent from the involved limbs. As the period of so-called shock disappears, the plantar reflexes first become positive, and then the deep reflexes reappear and finally become hyperactive.

The prognosis in intracerebral hemorrhage is

very poor, and over 75 per cent of the patients die during the first week of the illness. In the nonfatal cases, recovery may continue for months, and the end result will depend upon the amount of actual brain destruction that has occurred. Generally, in patients who recover, the ultimate prognosis for functional recovery is fairly good, and, with adequate application of rehabilitation procedures, these patients do very well.

Cerebral thrombosis. Intracerebral thrombosis is probably the most common type of vascular syndrome encountered in this age group. It may implicate any of the cerebral vessels, and the resulting symptoms will depend upon the vessel involved. The middle cerebral artery or its branches are most often implicated, resulting in contralateral motor weakness, hemihypesthesia, or speech disturbances involving the dominant hemisphere. Paralysis of the face and upper extremity is often more complete than that of the lower limb. If one of the smaller branches becomes occluded, only partial syndromes may occur.

The onset of symptoms is often slow over a period of hours or days and frequently occurs during sleep or rest when the blood pressure is decreased. There usually is no loss of consciousness, and headaches are infrequent. The spinal fluid is usually clear and the pressure normal. These patients may often give a history of mild transient episodes of vascular insufficiency for months or years preceding the occlusion.

The prognosis is good. Over 75 per cent of patients survive the acute episode even when severe. Treatment during the acute stage is symptomatic. The patient should be kept quiet during the first few days and given special attention to avoid pulmonary complications. Adequate nourishment should be maintained and urinary retention avoided. Immediate introduction of passive movement of the paralyzed limbs is important to avoid contracture. Active physical therapy is deferred until recovery begins. Anticoagulant therapy should not be used, since this type of treatment proves fatal if the patient has a hemorrhagic process. During the subacute stage, slight active motion should be encouraged while the patient is still in bed. The patient is raised on a back rest after seven to ten days and placed in a chair after about two weeks, depending upon the degree of involvement and the age of the patient. Older patients are activated more quickly. As function returns, the patient should be helped and encouraged to use the involved limbs by participating in a program designed to increase self-care and ambulation activities. The

process of rehabilitation of the stroke patient is an important part of the total treatment and must be instituted in each case. It generally can be carried out even in the home, provided the doctor understands his goal and is willing to spend a little time with the family and the patient in outlining and directing the course of therapy.

Basilar artery insufficiency. This relatively common vascular syndrome has been overlooked for many years. It occurs chiefly in older individuals and is characterized by transient episodes of neurologic manifestations that last for only a few minutes or hours at most. The predominate involvement is that of the cranial nerves and consists of short episodes of visual dimness, diplopia, ptosis, vertigo, and even dys-

phagia and dysarthria. The vertigo and visual disturbances predominate. Long-tract involvement may also occur and consists of a monoparesis or a hemiparesis which shifts from one side to the other. Transient episodes of sensory disturbances or confusional states may also occur. The diagnosis is suggested by the motor involvement of alternate sides of the body accompanied by the transient cranial nerve palsies.

Treatment consists of the immediate institution of anticoagulant therapy, which must be continued indefinitely. Even with this type of therapy, the prognosis is often guarded. Complete occlusion of the basilar artery results in a catastrophic disturbance with quadriplegia, dysphagia, and coma, which are often permanent.

PERICARDIAL PEDICLE GRAFTS may be satisfactory for repairing interauricular defects in human beings.

In dogs, pericardial pedicle grafts used in a fixed position to close ventricular septal defects and enlarge the outflow tract of the right ventricle show extensive tissue reaction and degeneration and are replaced with avascular connective tissue six months after operation. Similar grafts placed in the auricular septum survive without contracture.

The fate of pericardial pedicle grafts in a fixed position, as opposed to mobile grafts, depends to a large extent on pressures in the chambers in which the grafts are placed. Possibly, tissue transplanted to a cavity with a pressure higher than the capillary pressure of the graft is deprived of arterial supply and venous drainage, thus causing degeneration and subsequent scar formation.

D. LEPLEY, JR., P. F. HAUSMANN, and W. WEISEL: The fate of pericardial pedicle grafts used in a fixed position in various chambers of the dog heart. *J. Thoracic Surg.* 37:711-719, 1959.

GANGLIONIC BLOCKADE induced by hexamethonium administration lowers venous pressure of patients with congestive heart failure but does not significantly alter circulating blood volume. Thus, the level of venous pressure does not appear to depend only on the amount of circulating blood. Instead, the level of venomotor tone—the outpouring of impulses from the central nervous system—may be the most important determinant of the level of venous pressure.

Possibly, decrease in myocardial contractility produces a relative deficiency in the blood supply to the tissues which in turn initiates a reflex that causes increases in arterio- and venomotor tones. This central nervous system reflex, which is similar to that seen in hemorrhage and shock, presumably acts in a compensatory manner up to a point, beyond which it adds to the burden on the heart.

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How to Avoid Use of the Retrospectroscope in Coronary Occlusions

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IN THE NATURAL HISTORY of many diseases, certain complications are relegated to the level of unfortunate but untreatable components. The disease itself seldom changes but the knowledge that the physician acquires may change; this knowledge will help to treat the complication.

The purpose of this paper is to restate one of the more frustrating complications of coronary occlusion and to remind one of the specific medication to correct it. A case report is outlined.

THE PROBLEM

The complication is best described as a prolonged and febrile convalescence. This may be accompanied by moderate or severe anterior chest pain, congestive failure, effusions, and elevations of the sedimentation rate and white blood counts. The condition recurs.

Most patients recover but those who die are found to show pleural or pericardial effusion or both and large, flabby hearts but no second occlusion. The concept of a severe occlusion with complications leading to slow recovery or death was accepted until recently.

In the last few years, however, a more specific syndrome has been observed, and, what is better, a specific treatment to aid recovery has been found.

SPECIFIC SYMPTOMS

Dressler¹ reported 20 cases from 1955 to 1956 in which the patients suffered coronary occlusions followed in one to four weeks by a set of findings and symptoms. These findings included a rise in temperature to 102 to 103° F. and then a fall ranging from 99 to 100° F., a pericardial friction rub, pleural or pericardial effusion or both, and a persistent bilateral chest pain aggravated by body shifting and respirations. He called these symptoms the postmyocardial-infarction syndrome.

The same syndrome was reported by Ito and

associates² in postoperative pericardiotomy cases. It was observed only in those in whom wide incisions of the pericardium were made during the surgery and not in those who underwent vascular repairs near the heart. It occurred in rheumatic and nonrheumatic heart cases.

Pericardial rub following coronary occlusions has been reported since 1929.³ However, the friction rub of the postmyocardial-infarction syndrome occurs in many more patients than the 20 per cent listed by Friedberg.¹ Dressler¹ heard the rub in 80 per cent of his original series with a high proportion of the rubs being accompanied by pericardial effusion. He originally described cases of idiopathic recurrent pericarditis⁵ in 1955. Several of these cases fit the syndrome under discussion. In retrospect, this may have prompted his investigations which led to the formulation of the syndrome. Dressler believes that the etiology of the condition lies in the necrosis of the myocardium which occurs after an occlusion or extensive cardiac surgery. The full syndrome may occur anywhere from one to four weeks after the occlusion. Some patients may have 1 to 6 relapses of the syndrome following the original occlusion.

It is the recurrent nature of the postmyocardial-infarction syndrome that produces anxiety, depression, and excess hospital care for the patient and frustration for the physician. Blood cultures, urine cultures, a search for abscesses, and repeated electrocardiograms are of no avail. Antibiotics are useless against the symptoms.

TREATMENT

Any physician treating coronary occlusions must be alert for the development of the postmyocardial-infarction syndrome. In addition to being able to save the patient added concern about his "new" heart attack, the syndrome is rapidly cleared by the use of steroids. The length of time treatment is required varies from ten days to a month, depending on the relief of pain, the reduction of fever, and the decrease of the sedi-

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TABLE 1

	Day 1	Day 15	Day 29	Day 30	Day 45	Day 59	Day 74
Sedimentation rate	7 mm.	72 mm.	54 mm.	71 mm.	20 mm.	55 mm.	34 mm.
White blood count	17,500	11,200				17,000	10,000
Pericardial effusion		+++		0		+++	0
Friction rub	Days 2 to 9	Days 19 to 22					
Temperature range	Days 1 to 9 101° F. 100° F. 99° F.		Days 10 to 29 100° F. 99° F.	Days 30 to 45 98° F.	Days 60 to 70 100° F. 99° F.		

STEROIDS BEGUN

mentation rate. The usual precautions of dosage and gradual reduction in the amount of steroids must be followed. Dressler¹ feels that anticoagulants are contraindicated in view of the hemorrhagic pericarditis which has been observed at times in the syndrome. I have observed 1 patient treated with a combination of anticoagulants and steroids without ill effect. The following case is that of a patient in whom anticoagulants were stopped while he was on steroid treatment.

CASE REPORT

A 44-year-old white man had a coronary occlusion in January 1959 and a second such attack on February 1, 1960. A postmyocardial-infarction syndrome occurred thirty days later and recurred again fifty-four days after the occlusion. A retrospectroscope was employed at this point, and steroids were started.

Table 1 shows the rise and fall of the temperature, the sedimentation rate, the white blood count, and the coming and going of the pericardial effusion and friction

rub. Since the steroids were started, the patient has been free of symptoms for two months. Steroids were discontinued after three weeks.

Perhaps this article will prevent the use of hindsight and the retrospectroscope in overtreating patients with this syndrome and shorten the condition by the early recognition of the group of symptoms and the institution of the steroids.

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HYPOTHERMIA PARTIALLY PROTECTS the canine myocardium arrested by use of potassium citrate. A study at St. Louis University found that a canine heart stopped for thirty minutes with potassium citrate at 28° C. can perform more than 15 grammeters of stroke work in response to trial transfusion after arrest. In contrast, the heart of a comparable dog stopped at 37° C. is unable to exceed 8 grammeters of stroke work during the period after arrest.

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Glaucoma

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WITHIN THE PAST YEAR, a picture story in *Life* magazine, an excellent article in *Scientific American* magazine, several mentions in newspaper medical columns, such as that written by Dr. Alvarez, and other publicity have contributed to a gradually increasing awareness and interest on the part of the public in the problem of glaucoma. The awareness and interest of the medical family must not lag behind.

Glaucoma has been with us always, but its importance in producing morbidity and disability is steadily increasing. This is to be expected, since glaucoma, with some notable exceptions, is a disease of individuals beyond middle age. The incidence rises sharply in adults over 45 years of age. Today, more people are living longer, and, consequently, exposure to the hazard of glaucoma as to all other degenerative diseases is greater by far than in the past. We can calculate that there now must be twice as many patients who have glaucoma than there were twenty years ago, and, because of better case-finding technics, more publicity, and more available ophthalmologists, probably three times as many cases are recognized. However, it is certainly true that at any given moment only a fraction of all individuals with glaucoma have been found and are under treatment.

Various surveys in this country to determine the frequency of glaucoma, including an excellent one by Dr. Bruce Kantar at the University of Minnesota Hospital, result in the conclusion that about 2 per cent of adults have glaucoma. This significantly large figure plus the fact that over 15 per cent of all blindness is caused by glaucoma points to the presence of a major public health problem.

The ophthalmologist must, of course, recognize and accept his primary responsibility in the detection and treatment of glaucoma, but he needs greatly the interest and help of his colleagues in any field of medicine in case finding. Probably the general practitioner and internist will be the most help to him.

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Since the early stages of glaucoma, especially of the type usually termed chronic simple glaucoma, are gradual, insidious, and virtually without symptoms, the problem of case finding is both simplified and complicated. This job cannot be done by taking a good history, carefully examining the external aspect of the eyes, or even by observing the fundi with the ophthalmoscope. By the time obvious cupping of the disk can be seen, much damage has been done and the treatment has been made much more difficult. No reversal of this damage can be expected even with good control of the intraocular pressure. There are other means, such as careful studies of the visual fields, which are excellent but hardly applicable in widespread fashion because they are so time-consuming.

Routine tonometry is the only method of screening for glaucoma that is practical and reliable. No one will claim that tonometry is infallible. Even though, by definition, glaucoma is a condition in which the intraocular pressure is above normal, there are patients with normal pressures who have glaucoma. Paradoxical? Yes. It so happens that there is in the normal eye, and to an even greater extent in a few glaucomatous eyes, a diurnal variation which may result in a recording of normal pressure in the doctor's office at 3 P.M. and a definitely elevated pressure at 5:30 A.M., which is never discovered. In our present discussion, such cases must be omitted; fortunately, they are few.

Routine tonometry is easily performed, and the procedure can be quick, painless, and accurate within very satisfactory limits. For those who have not used it, the necessary equipment and the technic are here described.

EQUIPMENT

Tonometers are available from all supply houses. It is important that the accuracy of the instrument is certified by a tonometer testing station sponsored by the American Academy of Ophthalmology. Excellent tonometers are available for about \$65. Although there are other makes and types, the Schiøtz tonometer is almost universally used. The only other equipment neces-

sary in tonometry is a topical anesthetic, such as $\frac{1}{2}$ per cent Pontocaine (tetracaine) or Dorsacaine. These are now dispensed factory-packaged in plastic, dropper-squeeze bottles which are very handy. A couch, table, or chair with headrest that tilts back is needed to position the patient.

TECHNIC

One drop in each eye twice, about one or two minutes apart, produces excellent surface anesthesia within another minute. The patient is told to look straight up at the ceiling with both eyes open. Often this is made easier if the patient holds his arm up and looks at his thumb, which

can be positioned directly over his eye. The tonometer is applied lightly to the cornea. It must not be canted to either side; any tipping may distort the readings. Reading the instrument and finding the corresponding tension from the accompanying graph is easy. The whole procedure takes only a few minutes.

Any disease condition which threatens 2 per cent of our adult patients with misery, unhappiness, disability, and economic handicap cannot be lightly disregarded and deserves our most earnest attention. The detection of glaucoma can be easy. We must promote and practice the routine use of the tonometer.

SIMULTANEOUS INTRACUTANEOUS TESTING with standard mammalian tuberculin (PPD) and with antigens prepared from other types of mycobacteria may indicate whether tuberculin sensitivity is due to infection with virulent bacilli or with cross-reacting organisms.

In experimentally infected guinea pigs and patients in tuberculosis hospitals, tests with PPD and an antigen made from the Battey mycobacterium usually permit accurate differentiation of tuberculous infection from that with organisms of the Battey type. The homologous antigen generally produces a larger area of induration than the heterologous one.

Frequency of cross reactions to tuberculin in man varies geographically. In areas where cross reactions are common, the tuberculin test alone is seldom reliable for diagnosis.

In relation to the decreasing incidence of tuberculosis, infections with cross-reacting mycobacteria are becoming more frequent. The Battey organism, a nonphotochromogenic acid-fast bacillus, apparently infects and sensitizes many persons and rarely causes evident disease.

C. E. PALMER, L. B. EDWARDS, L. HOPWOOD, and P. Q. EDWARDS: Experimental and epidemiologic basis for the interpretation of tuberculin sensitivity. *J. Pediat.* 55:413-429, 1959.

GASTRIC ACIDITY apparently is necessary for acute multiple ulceration.

Continuous microscopic observation of the stomach wall and biopsy at various phases of histamine-induced ulceration in guinea pigs reveal 4 stages in the ulcerative process: [1] focal ischemia of the mucosa, with focal vasospasm of capillaries and arterioles; [2] flow of blood into the vessels of the superficial mucosa followed by complete stasis; [3] restoration of rapid blood flow into the ischemic area, causing rupture of damaged capillaries and hemorrhage; and [4] ulceration due to sloughing of hemorrhagic necrotic tissue.

Focal ischemic necrosis of the mucosal cells is often the initial lesion, but this change does not occur in tissues bathed in neutral or alkaline gastric juice. Some temporary metabolic disturbance may alter the permeability of the mucosal cells to allow entrance of acid gastric juice. The focal ischemia, then, is secondary to irritation by acid.

J. WATT: The mechanism of histamine ulceration in the guinea pig. *Gastroenterology* 37:741-759, 1959.

Bronchography in Cavitory Pulmonary Tuberculosis

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IN THE MANAGEMENT of every patient with active, pulmonary tuberculosis, 5 modes of treatment must be considered: (1) isolation and hospitalization during early phases of treatment; (2) adequate clinical evaluation, including accurate appraisal of roentgenographic residuals of tuberculosis; (3) specific antituberculosis chemotherapy; (4) surgical intervention when indicated; and (5) prognostic implications and recommendations to the patient.

Of all these considerations, the most important is that of clinical evaluation and, most specifically, accurate interpretation of roentgenographic findings. All other plans of management are eventually subservient to the interpretation of roentgenograms and an opinion regarding the nature of the pulmonary residuals after six or more months of chemotherapy.

If cavitory lesions persist, duration of hospitalization and isolation may be prolonged. Likewise, the need for surgical resection of persistent, open cavities must be considered. If cavities remain open and are not surgically resected, duration of chemotherapy and prognosis of the patient will be altered. When a cavity closes after six to twelve months of treatment, chemotherapy can be terminated at a specific time. On the other hand, in patients who have persistent, open cavities, antituberculosis chemotherapy is usually continued for an indefinite period of time.

We have noted that large, thin-walled, air-filled cavities may persist in the lungs of tuberculous patients after maximum radiographic resolution of all other parenchymal disease has occurred and roentgenograms have remained stable for many months. Some of these residual cavities may appear to have been closed when only conventional chest roentgenograms and planigrams are examined.

The principal aim of this study was to discover or better define thin-walled cavities through the use of bronchography. Endobronchial tuberculosis usually clears after several months of treatment. Residual, thin-walled cavities are then air-filled and frequently have a patent communicating bronchus. Adequate anesthesia, visual fluoroscopic filling with Dionosil in oil, immediate spot films, and postbronchographic films after four and twenty-four hours have been used in this study.

MATERIALS AND METHODS

All patients in this report were treated for at least five months before final evaluation by bronchography was attempted. Only cases with definitely visualized cavities at onset of treatment were considered. After five or more months of therapy and after the chest roentgenograms had become relatively stable, planigrams were made of the area of lung that had exhibited cavitation. Bronchography was performed in all cases in which pulmonary cavities seemed to have disappeared from conventional roentgenograms and from planigrams. In a few cases in which cavities appeared to persist on planigrams, bronchographic studies were conducted either to better define or to localize cavities.

Local anesthesia was accomplished by use of a 2 per cent Pontocaine spray plus repeated administrations of 2 to 3 cc. of 0.5 per cent Pontocaine into the trachea. A rubber catheter was then inserted into the trachea and the patient was placed on a fluoroscopic table that was equipped to take spot films. The rubber catheter was then filled with Dionosil in oil in order to make it opaque and more easily visualized on the fluoroscopic screen. By manual manipulation, the end of the catheter was then placed in either the right or left main bronchus, after which approximately 10 cc. of Dionosil in oil was injected through the catheter.

A tilting fluoroscopic table was used so that the contrast medium could be visualized as it

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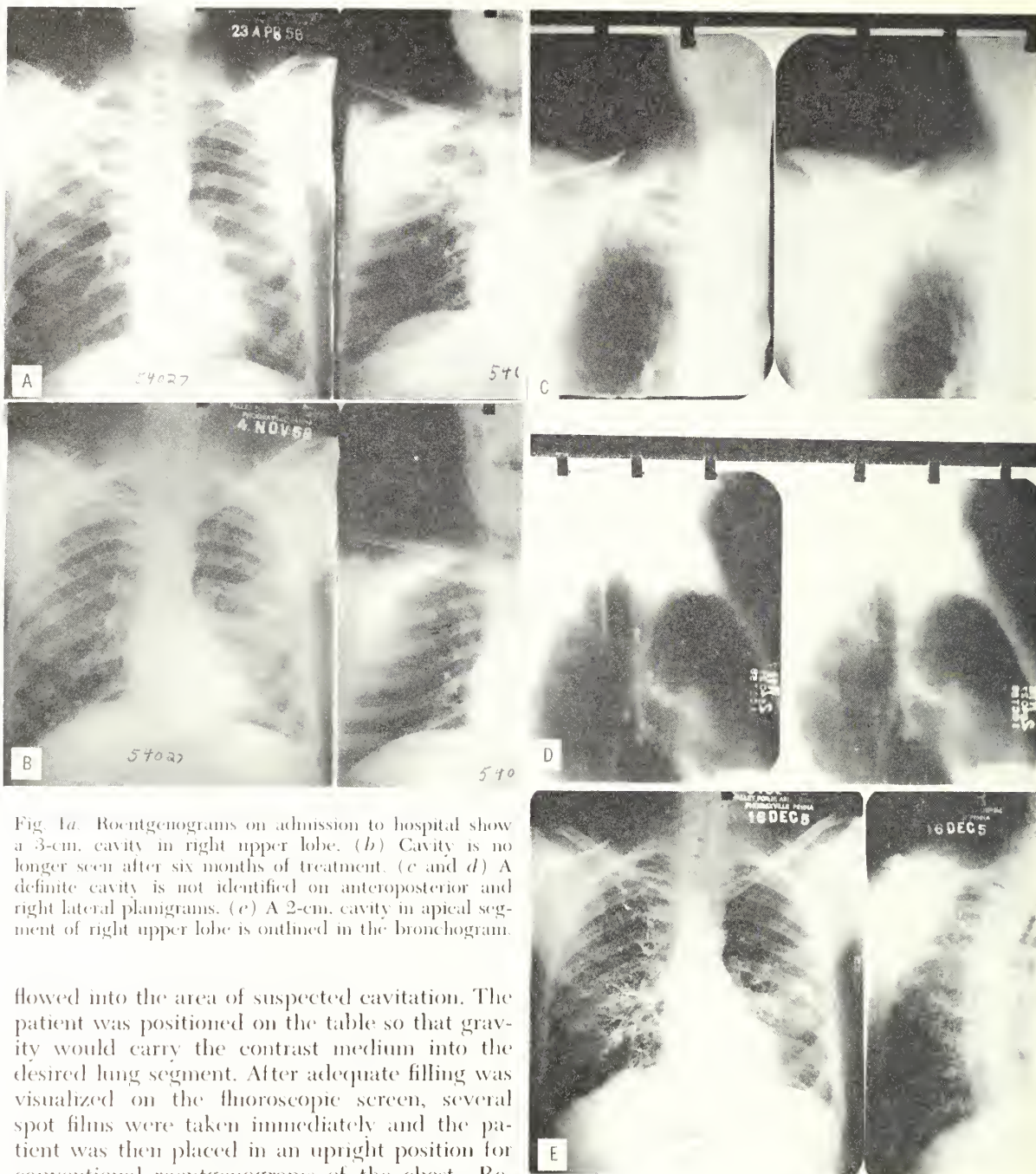


Fig. 1a. Roentgenograms on admission to hospital show a 3-cm. cavity in right upper lobe. (b) Cavity is no longer seen after six months of treatment. (c and d) A definite cavity is not identified on anteroposterior and right lateral planigrams. (e) A 2-cm. cavity in apical segment of right upper lobe is outlined in the bronchogram.

flowed into the area of suspected cavitation. The patient was positioned on the table so that gravity would carry the contrast medium into the desired lung segment. After adequate filling was visualized on the fluoroscopic screen, several spot films were taken immediately and the patient was then placed in an upright position for conventional roentgenograms of the chest. Repeat roentgenograms of the chest were then made four and twenty-four hours after instillation of Dionosil.

CASE REPORTS

Case 1. A 23-year-old man was admitted to the hospital with extensive tuberculosis throughout the right upper lobe. Gastric cultures were positive for mycobacterium tuberculosis. The chest roentgenogram revealed a 3-cm. cavity in the first right anterior interspace, which was seen best on apical films (figure 1a). Treatment consisted of the administration of streptomycin, 1 gm.

daily for ninety days; isoniazid, 16 mg. per kilogram of body weight daily; para-aminosalicylic acid (PAS), 12 gm. daily; and pyridoxine, 100 mg. daily. After six months of this therapy, moderate resolution of the disease in the right upper lobe occurred and only minimal, linear, and nodular densities were observed on postero-anterior and lordotic views (figure 1b). Planigrams taken through the right upper lobe in the anteroposterior projection did not reveal any definite evidence of cavitation (figure 1c). However, there appeared to be an oval radiolucency, which did not form a complete circle, at the anterior end of the first rib. Lateral planigrams

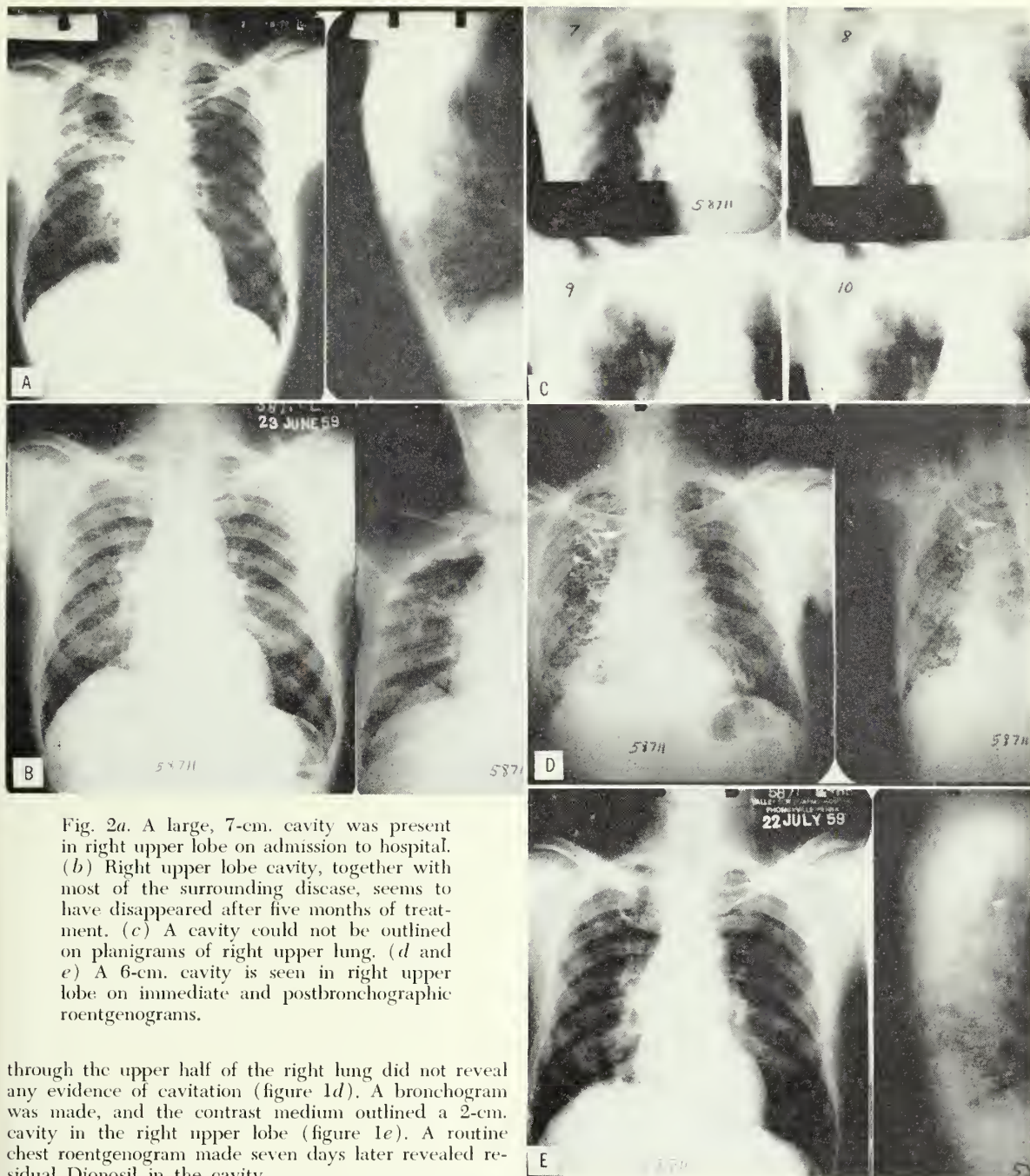


Fig. 2a. A large, 7-cm. cavity was present in right upper lobe on admission to hospital. (b) Right upper lobe cavity, together with most of the surrounding disease, seems to have disappeared after five months of treatment. (c) A cavity could not be outlined on planigrams of right upper lung. (d and e) A 6-cm. cavity is seen in right upper lobe on immediate and postbronchographic roentgenograms.

through the upper half of the right lung did not reveal any evidence of cavitation (figure 1d). A bronchogram was made, and the contrast medium outlined a 2-cm. cavity in the right upper lobe (figure 1e). A routine chest roentgenogram made seven days later revealed residual Dionosil in the cavity.

Six hours after one-third of the total daily dose of isoniazid and PAS was administered, the serum free isoniazid concentration was 1.2 to 2.4 $\mu\text{g.}$ per cubic centimeter. Cultures were positive for *M. tuberculosis* during the first three months of treatment, and the organisms recovered were completely sensitive to streptomycin, isoniazid, and PAS.

After eight months of chemotherapy, a right upper lobectomy was performed. The removed specimen revealed a cavity in the posterior segment of the right upper lobe. Scattered throughout the lining of the cavity were portions of necrotic debris, which were composed of palisaded histiocytes, epithelioid cells, and

scattered lymphocytes and multinucleated giant cells of the Langhans' type. Smears of caseous material and stained tissue sections did not reveal any acid-fast staining bacilli.

Case 2. A 58-year-old man was admitted to the hospital with far advanced pulmonary tuberculosis. The chest roentgenogram revealed a diffuse, nodular, and conglomerate infiltration in the right upper lobe and superior segment of the right lower lobe. One discrete nodule was seen in the left lung apex. A 7-cm. cavity was observed in the right upper lobe between the first and second anterior ribs (figure 2a). Chemotherapy con-

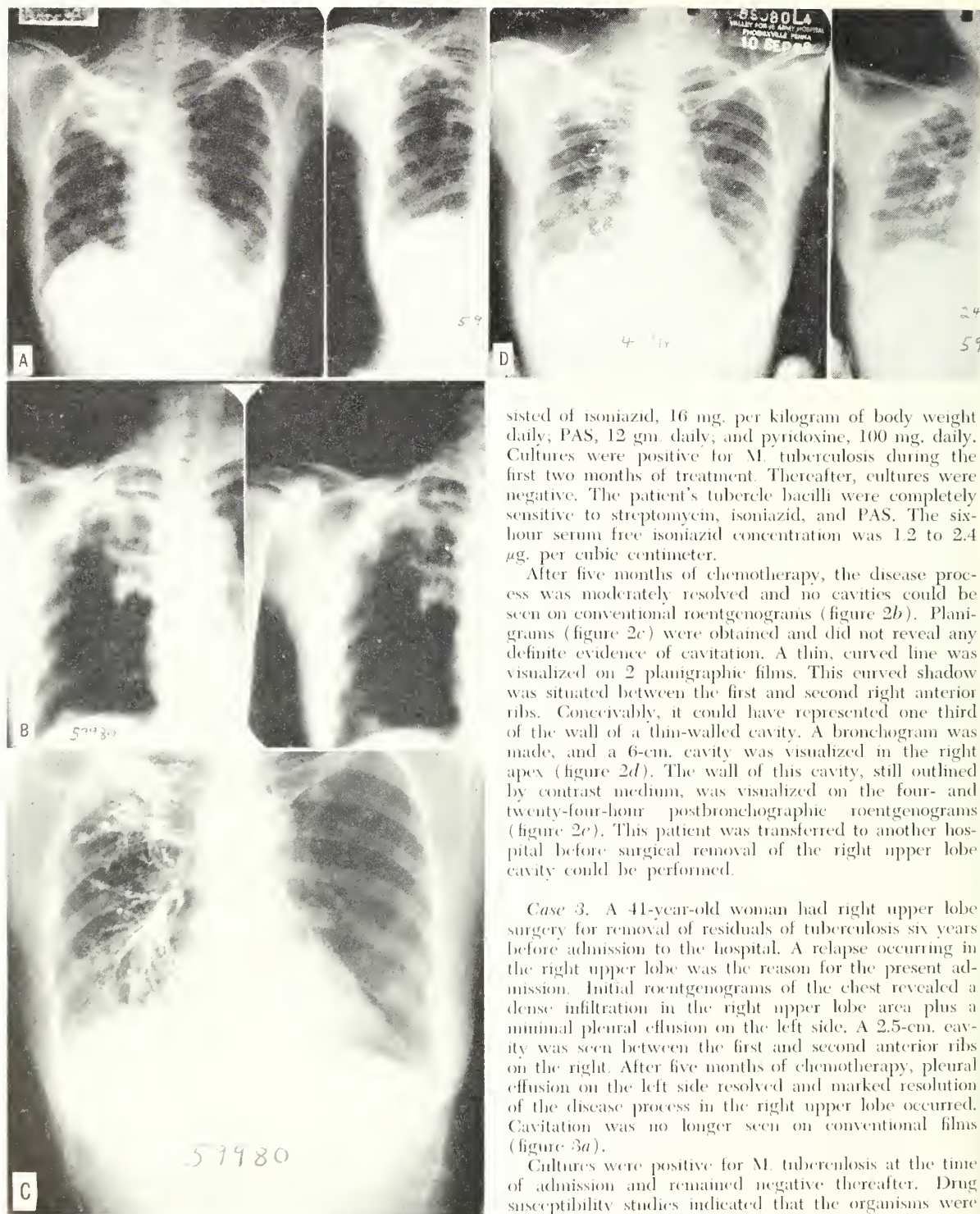


Fig. 3a. A 2.5-cm. cavity in right upper lobe seems to have disappeared after five months of treatment. (b) Planigrams reveal significant residual disease without definite cavitation. (c and d) A 3-cm. cavity in right upper lobe filled with Dionosil and was most clearly outlined on four- and twenty-four-hour postbronchographic films.

sisted of isoniazid, 16 mg. per kilogram of body weight daily; PAS, 12 gm. daily; and pyridoxine, 100 mg. daily. Cultures were positive for *M. tuberculosis* during the first two months of treatment. Thereafter, cultures were negative. The patient's tubercle bacilli were completely sensitive to streptomycin, isoniazid, and PAS. The six-hour serum free isoniazid concentration was 1.2 to 2.4 μ g. per cubic centimeter.

After five months of chemotherapy, the disease process was moderately resolved and no cavities could be seen on conventional roentgenograms (figure 2b). Planigrams (figure 2c) were obtained and did not reveal any definite evidence of cavitation. A thin, curved line was visualized on 2 planigraphic films. This curved shadow was situated between the first and second right anterior ribs. Conceivably, it could have represented one third of the wall of a thin-walled cavity. A bronchogram was made, and a 6-cm. cavity was visualized in the right apex (figure 2d). The wall of this cavity, still outlined by contrast medium, was visualized on the four- and twenty-four-hour postbronchographic roentgenograms (figure 2e). This patient was transferred to another hospital before surgical removal of the right upper lobe cavity could be performed.

Case 3. A 41-year-old woman had right upper lobe surgery for removal of residuals of tuberculosis six years before admission to the hospital. A relapse occurring in the right upper lobe was the reason for the present admission. Initial roentgenograms of the chest revealed a dense infiltration in the right upper lobe area plus a minimal pleural effusion on the left side. A 2.5-cm. cavity was seen between the first and second anterior ribs on the right. After five months of chemotherapy, pleural effusion on the left side resolved and marked resolution of the disease process in the right upper lobe occurred. Cavitation was no longer seen on conventional films (figure 3a).

Cultures were positive for *M. tuberculosis* at the time of admission and remained negative thereafter. Drug susceptibility studies indicated that the organisms were susceptible to streptomycin, isoniazid, and PAS. The six-hour serum free isoniazid concentration was 0.3 to 0.6 μ g. per cubic centimeter. The patient was treated with streptomycin, 1 gm. daily for ninety days; isoniazid, 16 mg. per kilogram of body weight daily; PAS, 12 gm. daily; and pyridoxine, 100 mg. daily.

After five months of therapy, planigrams were made through the right upper lobe (figure 3b) and no definite

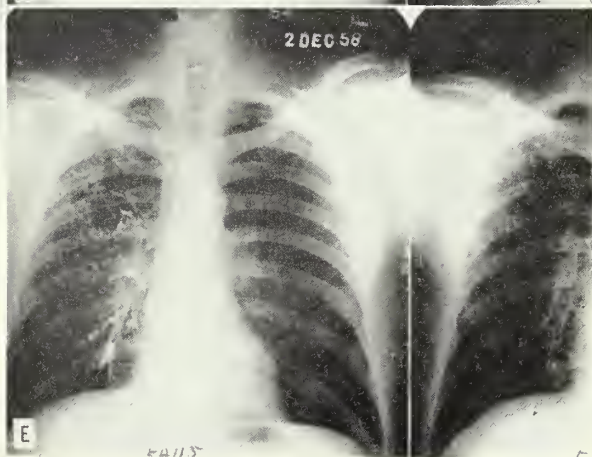
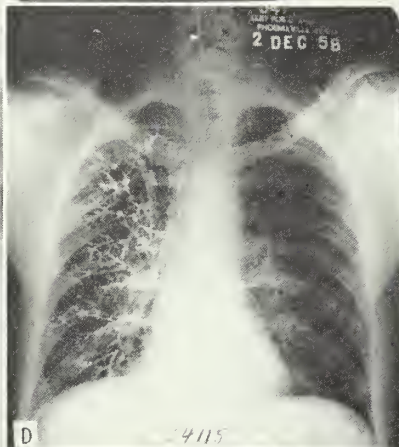
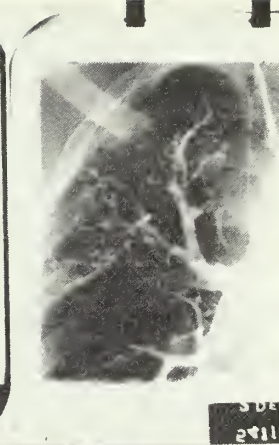
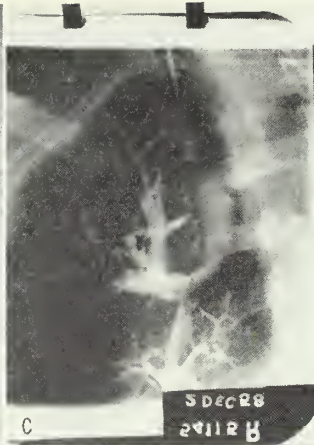
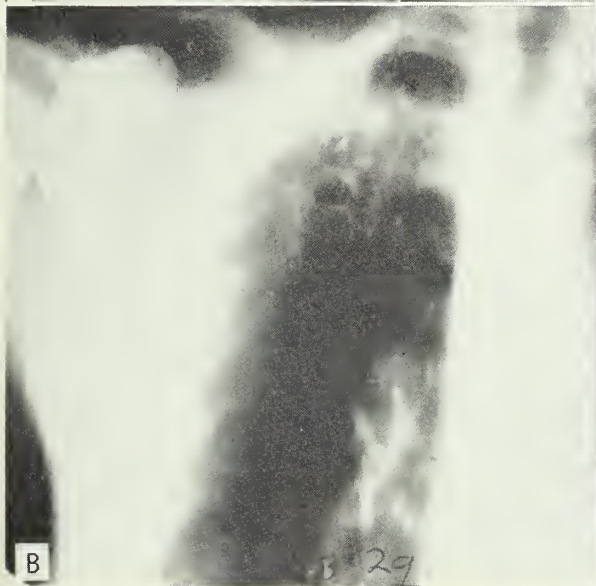
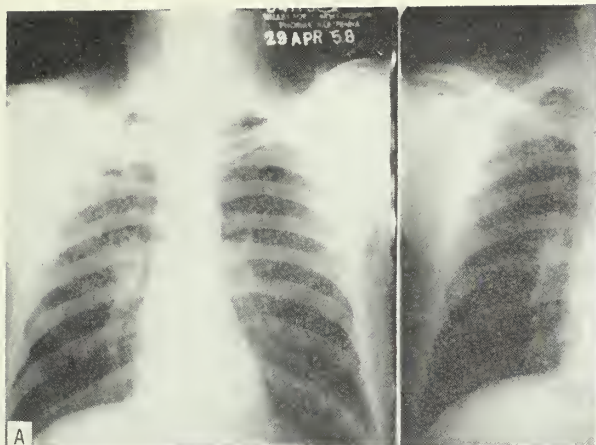


Fig. 4a and b. Although excellent resolution of disease in right upper lobe occurred after six months of treatment, a 2.5-cm. cavity is clearly seen on conventional roentgenograms and on planigrams. (c and d) Immediate spot films and upright bronchograms outline right upper lobe bronchi very well but the cavitory space did not fill. (e) A 2-cm. cavity is outlined by residual Dionosil on four- and twenty-four-hour postbronchographic roentgenograms.

cavity could be seen. A bronchogram was made (figure 3c) and revealed a well-outlined 3-cm. cavity with a fluid level that was created by the contrast medium. This cavity was in the right upper lobe at the site of the original cavity and was more clearly visualized on the four- and twenty-four-hour postbronchographic films (figure 3d).

The posterior segment of the right upper lobe was removed surgically, and a 4-cm. cavity was found. The lining of this cavity appeared regular, glistening, and grayish red. Microscopically, the wall of the cavity was composed of fibrous and hyalinized connective tissues.

In parts, there was granulation tissue with plasma cells, lymphocytes, and histiocytes. A bronchial communication was seen. No acid-fast staining bacilli were found in stained sections of the cavity wall or in smears of caseous material from other surrounding lesions in the adjacent lung. Cultures of removed lung tissue were negative for *M. tuberculosis*. From its gross and microscopic appearance, this cavity appeared to be open and healed.

Case 4. A 34-year-old man was admitted to the hospital with tuberculosis of the right lung. A chest roentgenogram revealed diffuse disease throughout the upper half of the right lung field, with a 2.5-cm. cavity in the

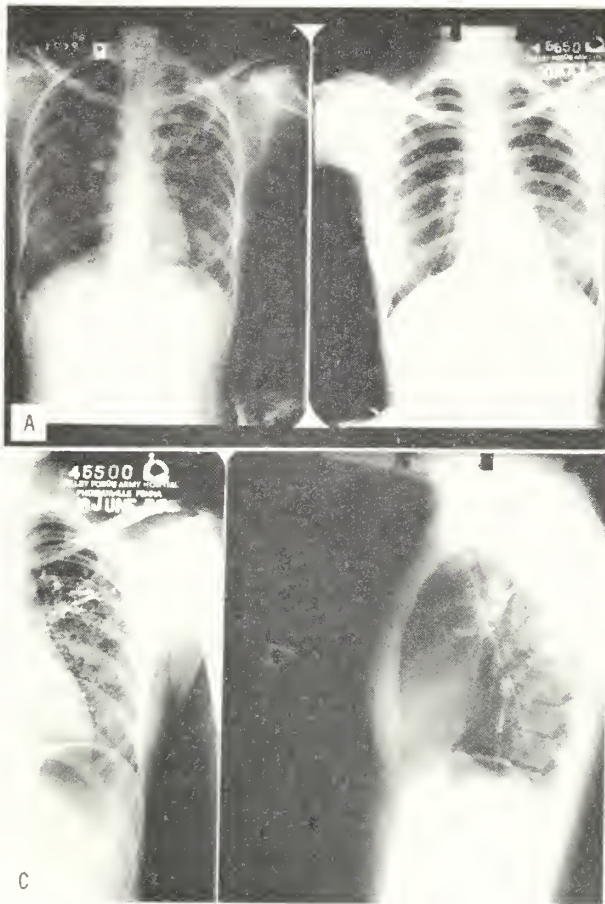
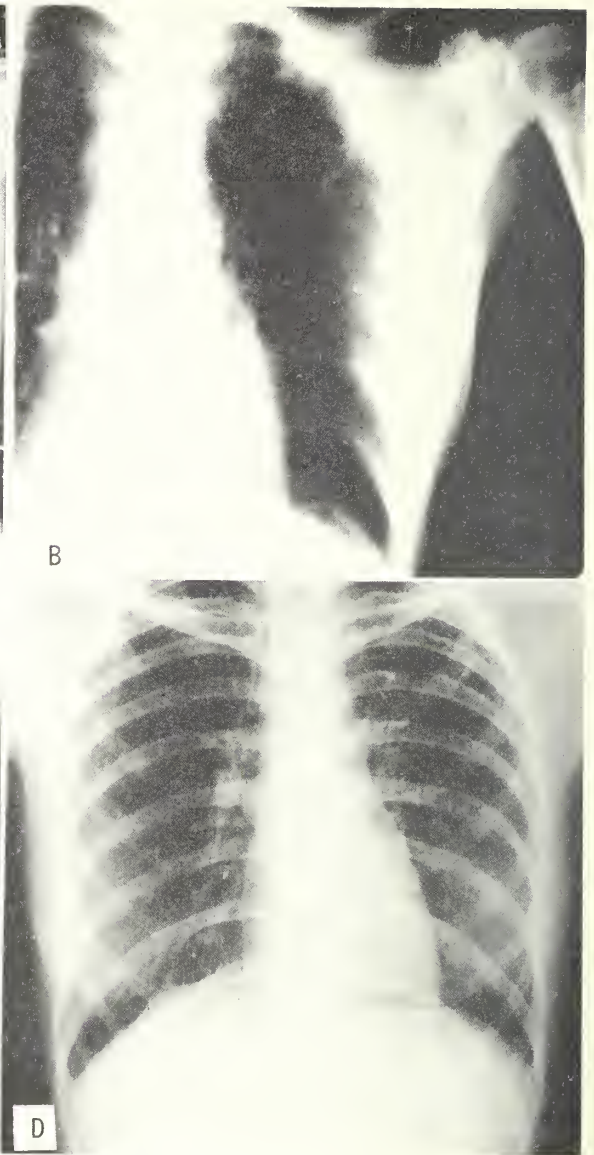


Fig. 5a. After seven months of treatment, a 5-cm. cavity seems to have disappeared from left upper lobe. Marked resolution of other areas of tuberculosis occurred. (b) Planigrams reveal medial and lateral curved lines in left upper lobe which did not form a complete circle. (c and d) Bronchographic and postbronchographic films reveal that a 7-cm. open cavity rather than a closed cyst remains in left upper lobe.

right first anterior interspace. After six months of chemotherapy, moderate resolution of the disease process occurred, but the cavity in the right upper lobe appeared to persist on conventional films (figure 4a). Cultures were positive for *M. tuberculosis* during the first two months of chemotherapy, and the organisms were completely sensitive to streptomycin, isoniazid, and PAS. Treatment consisted of isoniazid, 16 mg. per kilogram of body weight daily; PAS, 12 gm. daily; and pyridoxine, 100 mg. daily. Streptomycin, 1 gm. daily, was also administered for the first ninety days. The six-hour serum free isoniazid concentration was 1.2 to 2.4 μ g. per cubic centimeter. Planigrams made after six months of chemotherapy confirmed the presence of a cavity in the right upper lobe (figure 4b). A bronchogram made under fluoroscopic observation revealed good visualization of all segments of the right upper lobe; however, no evidence of cavitations was detected on immediate spot films (figure 4c). Upright bronchograms made several minutes later also failed to reveal any evidence of cavitation (figure 4d).



Postbronchographic roentgenograms made four and twenty-four hours later (figure 4e) revealed a 2-cm. cavity in the first right anterior interspace. This cavity was removed surgically with the right upper lobe, and its lining was found to be mostly hyalinized; however, there were some areas of active disease with inflammatory cells, epithelioid cells, and Langhans' giant cells. The surrounding lung contained fibrocaseous tuberculosis. Smears from fresh tissue were positive for tubercle bacilli. Cultures of the tissue were negative, but a guinea pig inoculation was positive for *M. tuberculosis*.

Case 5. A 22-year-old woman was admitted to the hospital, and the initial chest roentgenogram revealed a diffuse, scattered, nodular infiltration extending from the left apex to the fourth left anterior interspace. In addition, a 5-cm. cavity was visualized in the left lung apex. On the right side, an irregular, patchy shadow was observed in the second and third anterior interspaces.

After seven months of chemotherapy, marked clearing of abnormal roentgenographic shadows occurred (figure

5a). A minimal, linear, nodular, discrete residual shadow remained in the third right anterior interspace, and the disease on the left side seemed to have disappeared. The left upper lobe cavity appeared to have vanished completely. In retrospect, however, a 2-cm. curved line was visualized underlying the second left anterior rib. This small linear shadow, in all probability, represented a portion of the original cavity wall, but it was not detected until after the cavity was clearly outlined by bronchography. Planigrams (figure 5b) revealed semicircular, linear shadows in the medial and lateral portions of the left upper lobe. These shadows were not joined to form a complete cavity but did suggest the presence of an air-filled space.

A bronchogram (figure 5c) outlined most of the wall of a cavity in the left upper lobe with contrast medium. A repeat chest roentgenogram made six days later revealed Dionosil still outlining most of the wall of a 7-cm. cavity (figure 5d). This cavity was surgically removed with the apical posterior segment of the left upper lobe. The lining of this cavity wall was smooth and hyalinized and appeared to be healed except for a small portion near the bronchocavitary junction which appeared roughened. This small area revealed inflammatory cells, some epithelioid cells, and Langhans' giant cells. Smear, culture, and guinea pig inoculation from removed surgical tissue failed to reveal the presence of *M. tuberculosis*.

DISCUSSION

In cases 1, 2, and 3, there was excellent clearing of the disease process in the lung after five to seven months of chemotherapy. By examining conventional roentgenograms, it appeared that these 3 patients had sustained treatment results which would afford them an excellent prognosis. Cavities were present in the right upper lobe at onset of treatment in all 3 patients, and these cavities apparently subsequently disappeared. Planigrams were obtained in all 3 cases and revealed linear, nodular, and patchy areas of residual tuberculosis. No shadows definitely representative of thin-walled, well-defined, air-filled cavities could be seen on planigrams.

After bronchographic examination, large cavities were well outlined with contrast medium and, in cases 2 and 3, four- and twenty-four-hour postbronchographic films outlined the remaining areas of cavitation especially well. If bronchographic examination had not been performed in these 3 cases, it is reasonable to assume that chemotherapy would have been continued for approximately two years and then discontinued. The physician would have felt that these patients had made an excellent response to treatment and had minimal, noncavitary residuals remaining in the lung on conventional films and that the prognosis was excellent.

In case 4, a right upper lobe bronchogram was made despite the fact that a definite cavity appeared to remain in the right upper lobe after more than six months of therapy. This case was

included in order to demonstrate that air-filled cavities may not always be visualized on an adequate bronchogram unless postbronchographic roentgenograms are made. On the spot films taken immediately on the fluoroscopic table and upright bronchograms revealed that all 3 segments of the right upper lobe were filled with contrast medium. Although no cavity was detected on these immediate bronchograms, the lining of the remaining cavity was clearly outlined with contrast medium on the four- and twenty-four-hour postbronchographic films.

In case 5, a large, left upper lobe cavity plus its surrounding disease appeared to have vanished completely after seven months of treatment. A planigram of the left upper lobe revealed a semicircular line in the lateral portion and a less well-defined semicircular line in the medial portion. These shadows did not form a completely circumscribed thin-walled cavity but did suggest the possibility of a large residual cavity or cyst. A bronchogram revealed that Dionosil had entered this cavity and lined its wall. A repeat chest roentgenogram made six days after the bronchogram still revealed scattered Dionosil throughout the inner lining of this cavity. Had planigrams not been obtained, a giant cavity or emphysematous cyst in the left upper lobe might not have been suspected. Had bronchograms not been taken, the presence of a tuberculous cavity would not have been proved.

SUMMARY

Through proper use of bronchograms, it is possible to define pulmonary cavities that otherwise might not be detected. After adequate anesthesia and instillation of an intratracheal catheter, the technic of bronchography in this report includes direct fluoroscopic filling on a tilting table, immediate spot films, conventional upright films, and films made after four and twenty-four hours.

In 3 patients with cavitary tuberculosis, cavities seemed to have disappeared on conventional films and on planigrams after five to seven months of therapy. In all 3 cases, large air-filled cavities were demonstrated by bronchography. In a fourth case, a very adequate bronchogram failed to outline a residual cavity that was clearly demonstrated on postbronchographic films made after four and twenty-four hours. In a fifth case, a large, thin-walled cavity communicating with a bronchus was demonstrated by bronchography. This cavity was not seen on conventional roentgenograms. Planigrams demonstrated curved lines not forming a complete circle, which could have been misinterpreted as a large, closed, emphysematous cyst.

Mitral Commissurotomy

With Special Reference to Selection of Patients for Surgery

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MITRAL COMMISSUROTOMY has been applied extensively for palliation of mitral stenosis.¹⁻¹⁰ Over 1,000 such operations have been reported from a few surgical centers.^{8,10,11} The series of mitral commissurotomies herein reported is small by comparison. A review of this group was undertaken in order to examine critically our results and thereby to improve our own criteria for selection of patients for this procedure.

METHODS

A review was made of the charts of patients who had undergone mitral commissurotomy in this hospital during the eight-year period from December 1950 to December 1958. Operations were performed by various members of the chest surgery attending and resident staffs. Data concerning history, physical examination, roentgenographic study of the heart, cardiac catheterization, findings at operation, pathology, and autopsy reports were extracted from the charts. All electrocardiograms were reviewed by the authors. Right ventricular hypertrophy was diagnosed according to the criteria of Sokolow and Lyon.¹² Follow-up information was available on 59 of the 62 patients studied. Autopsies were performed on all but 1 of the patients who died, and the autopsy protocols were available for review. Of the surviving patients, 13 have undergone postoperative right heart catheteriza-

tion. Patients were classed as improved if they advanced one or more classes in the New York Heart Association functional classification.¹³

RESULTS AND INTERPRETATION

From December 1950 to December 1958, 62 patients were subjected to 63 closed mitral commissurotomies in the Veterans Administration Hospital; 58 were men and 4 were women. Their age distribution and preoperative functional classifications are shown in tables 1 and 2.

Of the patients, 40, or 65 per cent, had a history of acute rheumatic fever or chorea. This compares favorably with Wood's figure of 60 per cent.²

Predominant preoperative symptoms were divided conveniently into 4 categories: (1) fatigue, dyspnea, paroxysmal nocturnal dyspnea, or orthopnea; (2) right heart failure; (3) hemoptysis; and (4) systemic emboli. Their incidence is listed in table 3.

Systemic emboli were considered the only operative indication in the 2 class I patients and in 1 early class II patient who experienced only minimal respiratory symptoms with exertion. The 24 per cent figure for systemic emboli is comparable to the 28 per cent incidence in Olesen's series of 351 patients.¹⁴

Preoperative findings by auscultation of the heart are listed in table 4. Apical systolic murmurs are worthy of emphasis. Of the patients, 24, or 39 per cent, had apical systolic murmurs listed as grade I or grade II, while 13 patients, or 21 per cent, had grade III or louder murmurs preoperatively. In Olesen's group of mitral stenotic patients, 40 per cent had apical systolic murmurs.¹⁴

Preoperative fluoroscopic findings are listed in

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TABLE 1
AGE DISTRIBUTION

<i>Decades</i>	<i>Number of patients</i>	<i>Per cent</i>
3	9	14
4	34	55
5	14	23
6	3	5
7	2	3
Total	62	100

TABLE 2
PREOPERATIVE FUNCTIONAL CLASSIFICATION

<i>Class</i>	<i>Number of patients</i>	<i>Per cent</i>
I	2	3
II	34	55
III	22	35
IV	4	7
Total	62	100

TABLE 3
PREOPERATIVE SYMPTOMS

	<i>Number</i>	<i>Per cent</i>
Fatigue, dyspnea, paroxysmal nocturnal dyspnea, or orthopnea	59	95
Right heart failure	21	34
Hemoptysis	26	42
Systemic emboli	15	24

TABLE 4
PREOPERATIVE AUSCULTATORY FINDINGS

	<i>Number</i>	<i>Per cent</i>
Apical diastolic rumble	62	100
Apical diastolic thrill	15	24
P ₂ ↑ or P ₂ > A ₂	44	71
M ₁ ↑	30	48
Opening snap of mitral valve	15	24
Semilunar insufficiency murmur	21	34
Aortic systolic murmur	3	5
Apical systolic murmur	37	60

TABLE 5
PREOPERATIVE CARDIAC FLUOROSCOPY

	<i>Number</i>	<i>Per cent</i>
Left atrial enlargement	60	98
Enlarged pulmonary artery	29	48
Right ventricular enlargement	29	48
Left ventricular enlargement	21	34
Calcium in mitral valve	33	54

table 5. Approximately one-third of the patients had fluoroscopic evidence of some degree of left ventricular enlargement.

Table 6 lists the preoperative electrocardiographic findings. Included in the electrocardiograms considered normal were those with R-prime waves in V₁ or V₂ when the R-prime wave was smaller than the R wave. Almost one-quarter of these patients had electrocardiographic evidence of left ventricular disease and/or digitalis effect.

Preoperative catheterization data were available on 54 patients. Table 7 lists the results.

It is interesting to note that 34, or 65 per cent, of the 52 patients with pulmonary hypertension had accentuated pulmonary second sounds by auscultation, and 16, or 31 per cent, had a murmur of semilunar insufficiency.

Table 8 lists the cardiac findings at operation on 61 patients. Approximately one-half of the patients in this series had a regurgitant jet palpated by the operating surgeon. In addition, the operating surgeon reported that a regurgitant jet was produced or an existing jet was made more pronounced in 12 patients after fracture of the valve.

Pathologic interpretations were available on biopsies of left atrial appendages in 51 patients. Of the biopsies, 36, or 71 per cent, were normal and 13, or 25 per cent, showed subendocardial fibrosis. Evidence of active rheumatic lesions in the form of Aschoff bodies were noted in only 2 instances, or 4 per cent. These were the only 2 patients in the series suspected clinically of having an active rheumatic process, as evidenced by fever, increased sedimentation rate, anemia, and changing electrocardiograms. This incidence contrasts rather sharply with the experience of Decker and associates,¹⁵ who reported Aschoff bodies in 45 per cent of 183 left atrial appendages studied. McNecly and associates¹⁶ found no clinical correlation between Aschoff bodies and the usual manifestations of active rheumatic fever in the same 183 cases.

Follow-up information was available on 59 patients. Table 9 presents this information according to preoperative functional class. Follow-up time varied from three months to eight years, the average being twenty-three months.

A total of 15 deaths occurred in this series. Those taking place during the first thirty days after mitral commissurotomy were classed as operative deaths. There were 6 operative deaths, for a total operative mortality of 10 per cent and a divided mortality as follows: Class II 12 per cent, class III 5 per cent, and class IV 25 per cent. Table 10 describes in detail those pa-

tients who died and those who were unimproved by operation. Table 11 lists factors thought to contribute to death or poor operative results.

Most authorities writing on mitral commissurotomy caution against operating on people with acute, or at least active, rheumatic fever.^{14,17,18} In this series, 2 patients had clinical evidence, that is, fever, increased sedimentation rate, changing electrocardiogram, increased antistreptolysin O titer, and pathologic evidence, that is, Aschoff bodies, of rheumatic activity. Both patients were improved by operation and had uncomplicated postoperative courses.

Of the 4 patients who had postoperative illnesses compatible with the so-called postcommissurotomy syndrome, all had good, rapid responses to bed rest, salicylates, and penicillin. This 6 per cent incidence is in sharp contrast to that reported by Dresdale and associates,¹⁹ who found 33 instances, or 39 per cent, of the postcommissurotomy syndrome in 84 patients. Most of their patients responded well to adrenocorticosteroids, and they recommended routine preoperative administration of steroids.

Systemic emboli occurred in 4 patients, or 6 per cent, at operation. This incidence is comparable to that reported by Ellis and associates.²⁰ Of these 4 patients, only 1 had atrial fibrillation.

As noted previously, left atrial thrombi were present at operation in 9 patients. No correlation between a left atrial thrombus at operation and preoperative history of systemic embolization was apparent. Similarly, preoperative administration of anticoagulants did not significantly affect the incidence of left atrial thrombi.

The problems of refusion and secondary stenosis have not been mentioned. In this series, 1 patient required reoperation because of continued evidence of mitral stenosis. A second patient, who also had continuing evidence of mitral stenosis, died before reoperation was attempted; an autopsy was not performed. Both patients had suggestive evidence of continued rheumatic activity in the postoperative period. Glover and associates⁶ found no evidence of restenosis in 31 late postoperative deaths in 600 patients, and Black and Harken⁹ feel that if refusion does occur, it must be extremely rare. They do mention, however, secondary stenosis, which is distal fusion of the chordae tendinae and papillary muscles. Often, this problem is overlooked at the time of commissurotomy and is another factor leading to poor operative results. No instance of secondary stenosis was described by the operating surgeons in this series.

TABLE 6
PREOPERATIVE ELECTROCARDIOGRAPHIC FINDINGS

	Number	Per cent
Atrial fibrillation	26	42
Right ventricular hypertrophy	13	21
Right ventricular strain	19	31
Combined ventricular strain	7	11
Left ventricular strain and/or digitalis effect	7	11
Nonspecific ST-T changes	5	8
Within normal limits or with positional changes only	15	24
Miscellaneous, including right bundle-branch block and 1° atrioventricular heart block	2	3

TABLE 7
PREOPERATIVE CATHETERIZATION DATA ON 54 PATIENTS

	Number	Per cent
Normal pulmonary artery pressure and elevated pulmonary capillary wedge pressure (resting)	2	4
Elevated pulmonary artery pressure (resting)		
PA systolic < 60 mm. Hg		
PA mean < 40 mm. Hg	30	56
PA systolic > 60 mm. Hg		
PA mean > 40 mm. Hg	22	41

TABLE 8
FINDINGS AT OPERATION ON 61 PATIENTS

	Number	Per cent
Enlarged pulmonary artery	50	82
Left ventricular diastolic thrill	47	77
Enlarged left atrium	40	67
Left atrial systolic thrill	5	8
Enlarged right ventricle	1	2
Enlarged left ventricle	3	5
Calcium in mitral leaflets	38	62
Left atrial thrombus	9	15
Aortic valve lesions	3	5
Mitral insufficiency		
Grade I or II	29	
Grade III or more	1	30
		49

TABLE 9
POSTOPERATIVE FOLLOW-UP BY CLASS IN 59 PATIENTS

Class	Improved	Unimproved	Expired
I		2 (100%)	
II	21 (68%)	5 (16%)	5 (16%)
III	13 (59%)	3 (14%)	6 (27%)
IV			4 (100%)
	34 (58%)	10 (17%)	15 (25%)

TABLE 10
CHART OF POOR RESULTS AND DEATHS

Patient	Decade age	Preop. classification	Date of operation (yr.)	Auscultation	X-ray	Electrocardiogram	Cath. data	Operative findings	Result	Comment
E.B.	3	II	56	MS	LAE PAE	AF RVS	PAM=20 PCW=13	MS	Died postop.	Renal infarct preop. Trauma to left common carotid artery at operation.
D.U.	3	IV	54	MS+MI	LAE RVE LVE PAE Ca++	Combined strain	PAM=59 PCW=27	MS MI	Died at op.	Ca++ embolus to brain. Had operation in 1950; MI noted, nothing done.
M.F.	5	II	56	MS	LAE PAE	AF RVS	PA=58/32 PAM=40 PCW=16	MS Atrial thrombus	Died postop.	PI preop. On anticoagulants. CVA at operation.
A.M.	4	III	54	MS+MI	PAE RVE LAE	Combined strain	PA=83/41 PAM=61 PCW=30	MS	Died 7 months postop.	Ventricular tear at op. Later ventricular aneurysm. Died at operative repair.
S.K.	5	III	55	MS+MI SLI	LAE Ca++	P mitral	PAM=36 PCW=18	MS 1+MI	Died 5 days postop.	Ca++ embolus right internal carotid artery.
L.J.	4	III	53	MS	PAE RVE LAE	RVS RVH	PA=65/33 PAM=43 PCW=30	MS	Died 6 months postop.	PI postop. MI produced after fracture. Slight AS at postmortem.
W.B.	5	III	57	MS=MI AS SLI	PAE LVE LAE RVE Ca++	AF RVH	PA=80/24 PAM=53 PCW=24	MS 2+MI	Died 6 weeks postop.	4+ MI postop. Severe CHF.
W.B.	5	IV	53	MS=MI	Giant LA LVE RVE	AF Combined strain	PA=56/28 PAM=33 PCW=27	MS	Died 1957	Class III for years, then class IV myocardial insufficiency.
E.D.	7	IV	52	MS	LAE RVE	AF RVS	RV=96/13	MS	Died 8 months postop.	Died of perforated jejunum. Improved to class II before death.
L.S.	4	III	53	MS=MI SLI	PAE Ca++ LAE RVE	Combined strain AF	PA=70/43 PCW=14	MS 2+MI	Died 1955	No improvement. Severe CHF. Probably insufficient fracture.
A.P.	5	IV	52	MS=MI SLI	LAE Ca++ RVE LVE	DIVC AF RVS RVH	PA=40/18	MS AI	Died 1959	Class IV since operation. Myocardial insufficiency. Lived 7 years.
J.S.	5	II	55	MS=MI	PAE Ca++ LAE	AF	PAM=22 PCW=15	MS 3+MI LA thrombus	Died 3 days postop.	Acute myocardial infarct at operation.
C.C.	4	II	53	MS=MI	PAE LVE LAE Ca++ RVE	LVS and/or Dig. ef. AF	PAM=34 PCW=25	MS 1+MI	Died at op.	Ventricular fibrillation.
T.D.	4	II	54	MS=MI	LAE RVE LVE	AF LVS	PA=38/20 PCW=31	MS 1+MI	Died 4 years postop.	MI increased after fracture. Died during reop. with heart-lung machine.
S.A.	4	III	58	MS=MI	LAE PAE LVE Ca++	AF Combined strain	PA=50/30 M=40	MS 2+MI Slight AS	Died 4 months postop.	MI increased after fracture. Died 3 days after open repair.
L.O.	4	III	53	MS=MI	PAE RVE LAE Ca++	RVS RVH	PAM=66 PCW=35	MS 2+MI	Improved initially, now class III	Increased MI after operation.
G.V.	5	III	58	MS=MI	PAE RVE LAE Ca++	RVS RVH	PA=74/33 PAM=44 PCW=30/24	MS 1+MI	No improvement	Increased MI after operation.
L.L.	3	III	55	MS SLI	LAE	WNL	PAM=30 PCW=22	MS AS	No improvement	AS not diagnosed preop.
J.V.	4	II	50	MS	LAE LVE	Incomp. RBBB	PA=63/20 PCW=21	MS	No improvement	Insufficient fracture, possibly had recurrent ARF.
H.R.	4	II	58	MS=MI	LAE Ca++ PAE LVE	AF NSSTT	PA=45/27 PAM=32	MS 1+MI	No improvement	Had preop. embolus with right hemiplegia and aphasia.
W.V.	5	II	57	MS=MI	LAE RVE	AF	Not done	Min. MS 1+MI	No improvement	Valve quite open; little done. Multiple emboli preop.
N.F.	4	II	52	MS=MI	LAE RVE	LVS and/or Dig. ef.	PA=55/30 PAM=42	MS	No improvement	MI produced at operation.
D.K.	3	II	58	MS	PAE LAE	AF NSSTT	PA=WNL PCWinc.	MS	No improvement	Had minimal symptoms. Emboli main indication for operation.
J.F.	5	I	53	MS SLI	PAE LAE Ca++	AF	PAM=20 PCW=13	MS	No symptoms	Emboli preop. only.
K.L.	5	I	54	MS=MI	LAE RVE LVE Ca++	AF NSSTT	Not done	MS 1+MI	No symptoms	Emboli preop. only.

MS—mitral stenosis, LAE—left atrial enlargement, LVE—left ventricular enlargement, MI—mitral insufficiency (graded 1+ to 4+), SLI—semilunar insufficiency, AS—aortic stenosis, RVE—right ventricular enlargement, PAE—enlarged pulmonary artery, Ca++—calcified mitral valve, AF—atrial fibrillation, RVS—right ventricular strain, RVH—right ventricular hypertrophy, DIVC—delayed intraventricular conduction, LVS—left ventricular strain, WNL—within normal limits, RBBB—right bundle-branch block, NSSTT nonspecific ST- and T-wave changes, PAM—pulmonary artery mean pressure in mm. Hg, PCW—pulmonary capillary wedge pressure in mm. Hg, AI—aortic insufficiency, PI—pulmonary infarct, CVA—cerebrovascular accident, CHF—congestive heart failure, ARF—acute rheumatic fever, op.—mitral commissurotomy, PA—pulmonary artery pressure in mm. Hg, RV—right ventricular pressure in mm. Hg, Dig. ef.—digitalis effect

TABLE 11
FACTORS CONTRIBUTING TO DEATH OR POOR
OPERATIVE RESULTS

	<i>Number of instances</i>
Increase in or production of mitral insufficiency	9
Systemic emboli at operation	4
Exuberant mitral valve calcium	2
Operative trauma to heart or great vessels	2
Wrong preoperative diagnosis	1
Myocardial insufficiency	4
Inadequate mitral commissurotomy or restenosis of valve	2
Aortic valve lesion	2

COMMENTS

The 10 per cent operative mortality in this series compares favorably with mortality figures observed with the first 100 mitral commissurotomies reported elsewhere.^{5,8,21} Surgeons with more experience report operative mortality rates that are as low as 1 per cent for patients in other than class IV status.^{8,20}

Postoperative improvement figures also are quite similar to those previously reported.^{5,7,20} These figures are in sharp contrast to the medically treated series reported by Grant,²² who recorded a 42 per cent mortality in ten years and 34 per cent of patients made worse, and by Olsen,¹⁴ who reported for 351 patients an average age at death of 47 years.

Ellis and Harken⁷ have listed certain factors which portend poor operative results. These include: (1) age over 40 years, (2) calcium in mitral valve, (3) atrial fibrillation, (4) preoperative mitral valve area greater than 1 sq. cm., (5) estimated postoperative mitral valve area less than 2.5 sq. cm., (6) pregnancy, (7) multivalvular involvement, and (8) moderate to marked mitral insufficiency.

They found a striking decrease in the percentage of patients improved after mitral commissurotomy as the number of above adverse factors increased. If none of the factors was present, 96 per cent of their patients improved postoperatively, while if 5 or more factors were present, only 50 per cent improved.

In our relatively small series, mitral regurgitation accompanying mitral stenosis was a most difficult problem, not only from the standpoint of estimating its severity but also in determining the advisability of closed mitral commissurotomy for these patients. Mitral regurgitation characteristically exhibits symptoms dominated by fatigue, apical systolic murmur, left ventricular

enlargement and left atrial systolic expansion by fluoroscopy, and evidence of left ventricular involvement on electrocardiograms.²³ In this series, 60 per cent of the patients had apical systolic murmurs, 34 per cent had some degree of left ventricular enlargement by fluoroscopy, and 30 per cent had evidence of left ventricular disease on electrocardiograms. None of the patients had evidence of other types of heart disease involving the left ventricle, that is, high blood pressure or arteriosclerotic heart disease.

Table 12 relates these various adverse factors to postoperative improvement and to the incidence of mitral regurgitation at operation.

Data from a small series of patients such as this must be interpreted with caution. However, as one might expect, these data do suggest that the more findings characteristic of mitral regurgitation present in any given patient with mitral stenosis, the more likely is the finding of a regurgitant jet at surgery and the less likely is improvement of the patient by operation to be expected. Similarly, advanced cardiac disease, even without mitral regurgitation, generally portends a poor surgical result.

Review of this series has helped us view mitral commissurotomy in its proper light—as a palliative operation for mitral stenosis. It does not make the valve normal, nor does it correct all of the organic changes in the heart and lungs produced by longstanding mitral block. Likewise, it does not correct the myocardial insufficiency frequently associated with rheumatic heart disease,²⁰ nor, of course, does it correct abnormalities present in other cardiac valves.

The referring physician has at his disposal adequate clinical signs—age, mitral valve cal-

TABLE 12
EFFECT OF ADVERSE FACTORS

	<i>Number</i>	<i>Per cent improved</i>	<i>Per cent with MI</i>
1. Patients with none of below	15	67	7
2. Age: under 40	43	65	49
over 40	19	42	47
3. Calcium in valve: with	33	58	70
without	29	55	24
4. Atrial fibrillation: absent	36	72	44
present	26	31	54
5. Apical systolic murmur	37	51	76
Grade I	8	75	63
Grade II	16	63	81
Grade III to IV	13	23	77
6. Left ventricular enlargement	24	52	57
7. Left ventricular strain and/or digitalis effect, combined strain or nonspecific ST-T changes	19	32	58
8. Patients with Nos. 5, 6, and 7	10	30	80
9. Patients with Nos. 3, 4, 5, 6, and 7	6	17	83

cium, atrial fibrillation, apical systolic murmur, left ventricular enlargement, and electrocardiographic changes—to separate those patients for whom low operative mortality and a high degree of improvement can be expected from those with less optimistic outcomes.

With the recent and the expected future improvement in open heart technics for mitral valve repair, good arguments can be forwarded for using them in all patients with mitral stenosis and associated mitral insufficiency.

SUMMARY

The results of 63 mitral commissurotomies for 62 patients have been reviewed. Operative mortality and improvement statistics are similar to those reported previously with the first 100 mitral commissurotomies. Careful attention to proper selection of patients for surgery and careful surgical technics have been and will continue to be the keys to over-all satisfactory results.

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STEROID THERAPY is effective in the prevention and control of intense inflammatory reactions to chemical and bacterial agents and surgical trauma in the respiratory tract. Bronchospasm also is inhibited by steroids.

Use of steroids is advisable before, during, or after intrathoracic operations for patients who [1] have asthma, emphysema, or poor pulmonary reserve; [2] are old and debilitated; or [3] have acute respiratory impairment due to, for instance, bronchospasm, bronchopneumonia, or infection resulting from spread of secretions into the nonoperated lung during anesthesia.

As a prophylactic measure, 10 mg. of prednisone or 8 mg. of methylprednisone is given four times a day thirty-six to forty-eight hours preoperatively. The night before operation and the next morning, 100 mg. of cortisone acetate is administered intramuscularly. Postoperatively, this amount is given twice daily until the patient is able to take steroids orally. The dose is discontinued gradually.

For acute respiratory distress during or after operation, 100 to 400 mg. of intravenous hydrocortisone is given immediately. A continuous drip, for a total dose of 200 mg., may be necessary during the next twenty-four hours.

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Clinical Findings in Aphasia

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Minneapolis

IN 1861, Paul Broca^{1,2} described two cases of loss of speech from cerebral injury and presented the autopsy findings before the Anatomical Society of Paris. He labeled both cases *aphemia*, which he defined as loss of the faculty of articulated speech without impairment of comprehension or loss of intelligence, and attributed the defect to lesions of the second and third frontal convolutions. He argued that it was more correct to localize faculties by convolutions of the brain than by protuberances of the skull, as phrenologists under Gall had tried to do.

Broca's first patient had been an epileptic since youth and had suffered a progressive onset of right hemiplegia, loss of speech, swallowing difficulty, and personality changes. The second patient was 84 years old and had been confined eight years in the infirmary at Bicetre for senile debility. Both patients were critically ill when examined by Broca, and both brains were extensively damaged. The brains were not cut but were preserved in the museum at Dupuytren. Many years later, they were reexamined by Pierre Marie,³ who pointed out that the superficiality of the clinical examination had permitted many symptoms to be overlooked and had allowed the obvious fallacy of associating isolated symptoms with circumscribed lesions in brains as deteriorated as these.

Nine years later, the researches of Fritsch and Hitzig in Germany and Ferrier in England established the existence of a motor cortex in the frontal lobe of various mammals.⁴ In 1874, Heschl traced auditory radiations to the temporal lobe, and Wernicke⁵ described sensory

aphasia, considered to result from temporal lobe lesions. During the same period, Munk and Schaefer identified the visual cortex in the occipital lobe.⁴

It was then reasoned that a center in the frontal lobe contained cells in which were stored images of movements for speaking words, one word in each cell, and another center in which movements for writing words were stored. In the temporal lobe were cells containing images of the sounds of words, and in the parietooccipital cortex were cells containing images of written and printed words. These centers were surrounded by functionless cortical territory that contained cells in which new words could be stored. All centers were connected by fiber tracts. This has been called the mosaic theory of cortical function. From this formulation, it was possible to deduce motor or sensory aphasia resulting from destruction of a center (central aphasia) or from destruction of a fiber tract (conduction aphasia). This led to a period in the history of aphasia which has since been known as the age of the diagram makers. Symptoms that should result from lesions at various sites were deduced, patients who exhibited these symptoms were sought, and autopsies were procured when possible. Wernicke⁵ was candid enough to state that few cases in the literature were adequately described, and cases he had seen did not support his views of conduction aphasia.

Two men of the period rejected the popular viewpoint, and their writings on aphasia were ignored for almost fifty years. Hughlings Jackson⁶ wrote that, in his experience, the lesions which produced aphasia could be found anywhere in the distribution of the middle cerebral artery and that to localize speech and to localize the damage that destroyed speech were two dif-

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ferent things. Freud,⁷ far ahead of his time, suggested that one think in terms of cortical fields rather than of centers and fiber tracts and in terms of dynamic processes rather than of static faculties contained in cells. This, he thought, was not incompatible with localization. He described the speech area as a field at the perimeters of which were motor and proprioceptive, acoustic, and visual areas. If one imagined a movable lesion within this field, the closer the lesion approached one of these primary areas, the more the observed symptoms could be expected to partake of its character.

Actually, almost the same view is expressed by Penfield and Roberts⁸ in their recent book, *Speech and Brain Mechanisms*, based on evidence obtained from a ten-year study of seizures, Amytal sodium injections, electrical stimulations of the cortex, cortical excisions, and post-surgical aphasia. They go beyond Freud in considering the primary auditory and visual areas and the primary and secondary somatic sensory areas in the brain not as "end-stations" but as transmitting areas, which send streams of impulses from the periphery and perhaps from other parts of the cortex to thalamic nuclei for integration. Similarly, the primary and secondary motor areas of the cortex are thought to be activated by streams of impulses from the thalamus, which is a part of that which they term the centrencephalic system.

It was not until 1926 that Henry Head,⁹ a pupil of Hughlings Jackson, constructed a series of tests for aphasia and systematically administered them to 26 aphasic patients with head injuries resulting from gunshot wounds incurred in World War I. Intensive clinical study of these patients led Head to consider aphasia a defect of symbolic formulation and expression. He recognized 4 clinical forms of the disorder:

1. *Verbal aphasia*, resulting from lesions of the pre- and postcentral convolutions, characterized by loss of articulated speech, with comprehension impaired but with rapid recovery.

2. *Syntactic aphasia*, resulting from lesions of the upper temporal convolution, characterized by jargon, slurred speech, impairment of rhythm, and phrasal memory.

3. *Nominal aphasia*, resulting from lesions of the angular gyrus, characterized by loss of naming and of comprehension of the meaning of words.

4. *Semantic aphasia*, resulting from lesions of the supramarginal gyrus, characterized by disturbance of comprehension of the significance of words and phrases as a whole.

It is significant that, with careful study of even

a small number of patients, the traditional dichotomy between motor and sensory aphasia began to disappear. Head's work was limited by the fact that only 26 patients were studied and that little was generally known of objective mental measurement in his time.

In 1935, Weisenberg and McBride¹⁰ published a study of 60 aphasic patients, using controls and standardized measurements. An average of fifteen hours was spent examining each patient. The study offered substantial evidence that both receptive and expressive processes were always disturbed in aphasia, and isolated disturbances of single modalities, such as agraphia, alexia, and acalculia, were not found clinically. Weisenberg and McBride classified the patients they observed as predominantly expressive, predominantly receptive, amnesic, or expressive-receptive. Although this study is a landmark in the history of aphasia, the system of classification adds little to its significance. If both receptive and expressive impairment are always present, the words are wastebasket terms in the context of aphasia. Actually, longitudinal studies are necessary in order to arrive at a stable classification system, or one runs the risk of confusing symptoms characteristic of various stages of recovery with types of aphasia.

Recently, Wepman and Jones¹¹ of Chicago University completed a factor analysis of test responses obtained from a sample of 168 aphasic patients. Their findings have not yet been published in final form, but preliminary reports indicate that the general ability of aphasic patients to manipulate verbal symbols is reduced, comprehension is impaired, and visual and motor factors also play a role.

Establishment of an aphasia center at the Veterans Administration Hospital in Minneapolis in 1948, as part of the neurology program, made it possible to study a large series of aphasic patients over extended periods of time. Since no method of examination existed which was comprehensive enough to yield sufficient information or sensitive enough to reflect changes that occurred with recovery, work was begun immediately on constructing a test. No assumptions were made about the nature of aphasia, and no terms were used which were not operationally defined. We set out to study as objectively as possible the ways in which aphasic performances differed from normal performances and the ways in which the performances of aphasic patients differed from one another. Obtained data were analyzed, and the test was revised each year until 1955 in order to eliminate artifacts and explore areas considered to

need further investigation as new insights were gained. In 1955, the sixth, or research, edition of the Minnesota Test for Differential Diagnosis of Aphasia was printed and made available to hospital and university clinics for experimental use. At present, a research grant from the Institute of Neurological Diseases and Blindness is permitting more extensive processing of data than was previously possible or than was indicated before the test itself was considered reasonably adequate. More than 550 aphasic patients have been studied here, although not all have been examined on the same form of the test.

NATURE OF LANGUAGE DEFICIT IN APHASIA

As a result of these studies, we consider it most meaningful to define aphasia as a general language deficit resulting from brain injury, which cuts across various language modalities, such as understanding, speaking, reading, and writing and upon which specific motor or perceptual deficits may or may not be superimposed.¹² This general language deficit is characterized by reduction of vocabulary and reduction of verbal retention span.

Reduction of vocabulary. This can be observed in all language modalities. The Ammons Full-Range Picture Vocabulary Test is a standardized test for auditory comprehension of vocabulary. Speaking vocabulary can be tested by asking the patient to name objects or, if he can do this, by use of a standardized vocabulary test, such as is found on the Binet and Wechsler scales. There are, of course, standardized tests for reading and spelling vocabularies. Aphasic patients characteristically show reduction on all these scales before, and improvement after, treatment. In general, comprehension and reading vocabularies, involving recognition only, exceed speech and spelling vocabularies, which also involve recall, but the differences are not large.

Loss of vocabulary may be almost complete or may be so slight as to be manifest only in the occasional loss or misuse of a word, such as everyone experiences at times. In the aphasic patient, this occurs more often, and the reduction of vocabulary is characterized by certain regularities. First, the words that are best retained and first recovered are the most frequently used words in the language. Second, as vocabulary is recovered, words that are associated in meaning are confused, such as chair and table, boy and girl, green and red, Monday and Tuesday, June and July, and read and write.¹³ The same kinds of errors appear whether the pa-

tient is asked to point to objects as they are named, to name objects, or to read or write the word. The errors are not specific to any one modality.

Reduction of verbal retention span. In aphasia, errors tend to increase as the stimulus, or even the response, increases in length. For example, a patient may be able to point to objects named singly but may make errors if he is asked to point to 2 or 3 of the same objects named in a series. Many patients can follow short directions but not 2 or 3 of the same directions combined. Some patients can repeat sentences of 3 or 4 words but not sentences of 5 or 6 words. Like reduction of vocabulary, reduction of verbal retention span also cuts across modalities. A patient may be able to tell 2 things that a good citizen should do but not 3 because, by this time, he has forgotten what he was asked or what he has just said. In such a case, he may follow some tangential association and appear exceedingly confused. Reading comprehension is affected because, by the time the patient has reached the end of a sentence or paragraph, he has forgotten what it said at the beginning. Many patients are able to write single words to dictation but cannot write a sentence of 4 or 5 of the same words dictated as a unit. Spontaneous writing commonly reflects the defects found in speech.¹⁴

The finding of a language deficit present in all cases of aphasia, present in all language modalities, and statistically verified by the scalability of aphasic responses to test items, which have been reported in the literature¹² but which I shall not discuss here, is, of course, wholly incompatible with theories of aphasia based on topographic representation of functions in the cortex or with theories based on sensory or motor dichotomies.

However, reduction of vocabulary and reduction of verbal retention span are not the only symptoms found in aphasia but are merely deficits common to all patients and at all severity levels. In the next section, we shall return to the idea of Freud's movable lesion and consider what symptoms may be superimposed with the involvement of specific cerebral processes.

IMPAIRMENT OF AUDITORY PROCESSES

It has been stated that auditory processes are always impaired in aphasia. All clinical studies in the literature are in agreement about this. Jackson¹⁵ considered the arrangements underlying speech to be auditoarticulatory arrangements. Freud⁷ considered the associative activity of the acoustic element to be the central part of

the whole speech function. We learn language by hearing it. Young deaf children do not acquire language unless taught through other modalities. In a recent normative study, Templin¹⁶ found that, by the age of 3, children have acquired all the structural forms of adult speech—and all by ear. Fairbanks¹⁷ and Fairbanks and Guttman¹⁸ consider the speech apparatus a servo system that returns the output to a place of control for comparison and manipulation of output, and they have produced disintegration of speech patterns by experimentally delaying auditory feedback. Thus, it would appear reasonable to assume that much of the language disintegration in aphasia is secondary to impairment of auditory processes, and this has in fact been a fruitful field of investigation.

Street¹⁹ of this center studied hearing losses of 90 aphasic patients measured by pure-tone audiometric testing and found that they did not differ significantly from hearing losses of normal persons of similar age who had been exposed to similar conditions of noise. Simmons, formerly of this center, in a study not yet published, found no correlation between the degree of hearing loss in aphasic patients and the difficulty in understanding spoken language as measured by the Ammons test and others for comprehension in the aphasic battery. Impairment of auditory comprehension in aphasia, then, is not related to hearing loss. Aspects of auditory impairment in aphasia currently being studied are as follows:

Impaired auditory discrimination. The best test we have found for impairment of auditory discrimination in aphasic patients consists of a set of pictures of paired objects with names that sound alike, such as face-vase, goat-coat, chair-stair, rope-robe, card-cart, and so on. The patient is asked to point to the picture named by the examiner. Most aphasic patients tested so far have made some errors on this test; some have missed as many as 50 per cent of the items. If aphasic patients are asked to point to letters of the alphabet as they are named in random order, they are most commonly confused between letters that sound alike, such as e-d-e-g-p-t-v, a-i-k-h, i-v, and u-q. The evidence seems to indicate that, even when hearing acuity is unimpaired, some aphasic patients experience interference or distortion of sounds, producing defective discrimination to varying degrees. Some patients have recovered well enough to report that speech had previously sounded strange to them or that people did not seem to be talking correctly.

Impaired word recognition. A recent analysis

of errors made by 50 aphasic patients in recognizing words showed that, when the group was divided at the median, 28 per cent of errors of the severely aphasic group and only 12.5 per cent of errors of the mildly aphasic group were made by confusing words which sounded alike. On the other hand, 54 per cent of errors of the former group compared to 84 per cent of those of the latter group were made by confusing words associated in meaning, such as chair-table and knife-fork. There was no correlation between the two types of errors. We are therefore justified in considering impaired auditory discrimination and impaired auditory recognition of words as two distinct disturbances of auditory functions and in considering impairment of discrimination more serious. As the Ammons test results show, aphasic patients generally show some impairment of word recognition. If such impairment affects high frequency words, it has prognostic importance; otherwise, it is not significant.

In an early study, we classified 138 aphasic patients into 4 groups according to the amount of functional speech present at discharge:

1. *Excellent functional speech* was defined as the ability to discuss normally any subject the patient had previously been able to discuss.

2. *Good functional speech* was defined as speech normal in ordinary situations but defective under pressure or under demands for long or complex responses.

3. *Limited functional speech* was defined as the ability to make needs and wishes known in intelligible speech, although vocabulary was limited and responses defective.

4. *No functional speech* was defined as an inability to make correct voluntary responses, although there might be some residual language.

We then compared the initial performances of these 4 groups on tests for auditory comprehension and found that, on admission, patients in the 2 lower groups made more than twice as many errors on tests for auditory comprehension as patients in the 2 upper groups.²⁰ All of the 31 patients in the lowest group, who gained no functional speech although they received intensive treatment, made errors in pointing to common objects named by the examiner on initial testing. Of the 107 patients who regained some degree of functional speech, only 4 made errors on this test. Thus, we found a simple test with high prognostic value if patients are examined after they have stabilized physiologically. We are justified in considering that, if language processes are disrupted to the point where

a patient shows confusion in pointing to common objects named by the examiner, statistic probabilities are against recovery of speech. Exceptions are probably those cases in which lack of recognition was related to imperception.

Studies of auditory perception show that, if word frequency is held constant, long words are discriminated more easily than short words, which present fewer perceptual cues.⁸ Undoubtedly, this is one of the reasons why aphasic patients have difficulty with the so-called "little words" — the structural words of language, such as was, has, is, in, if, and of — which are even harder to discriminate in connected speech than in isolation, since they are unstressed in a sentence.

Impaired verbal retention span. This is a highly reversible condition present in all aphasic patients and so does not have prognostic importance. It has considerable clinical importance, however, for a verbal retention span can be increased measurably by intensive controlled auditory stimulation, and, as it increases, speech, reading, and writing tend to increase simultaneously. Treatment of aphasia has probably been improved more by this finding than by any other.

Impaired auditory recall. This is a construct rather than an empiric finding, although a good many observations support it. If one asks an aphasic patient with severe auditory impairment to name a set of pictures, one will probably get jargon responses, such as these to cigarette: leatherswak, easbizi, and spindeljug. If one asks the patient not to try to say the word but to listen as it is said, try to think it, and just let it come out when he has it, responses begin to sound more like the stimulus, such as these words for coffee: cofnik, soffee, offee. Eventually, the response is correct. If this kind of intensive and controlled stimulation is given on the same set of words every day for a week or two, the patient will be able to name most of them correctly by the end of the period. Other responses will be close approximations. Jargon responses will first decrease, then disappear. Mispronunciations are inconsistent and have no relation to the complexity of motor integration required to produce a given sound. The same sound will be pronounced correctly one time and erroneously another. Sounds requiring complex motor integrations will be substituted for sounds that are much easier to say, and combinations of consonants will be substituted for simple consonants.²⁰

We surmise that, as the lesion moves closer to the primary end-station of the acoustic nerve in the superior temporal gyrus, the more severely

auditory discrimination will be affected and that, when only a mild reduction of retention span is present, the lesion is at a greater distance from Wernicke's area.

IMPAIRMENT OF VISUAL PROCESSES

Visual impairment is present in some aphasic patients but not in others. One cannot infer that visual processes are impaired on the basis of even severe reduction of reading or writing ability, because reading and writing will reflect whatever impoverishment of language is present. Most patients with visual involvement can be shown to have a field cut; however, field cuts exist when no other visual deficit is present. The following deficits have been observed clinically:

Impairment of visual discrimination. Unless involvement is unusually severe, most aphasic patients are able to match pictures, colors, and simple geometric forms. However, patients with impaired visual discrimination tend to confuse letters with similar visual configurations, such as b-d-p-q, h-n-r-u, f-t, and w-m in lower case print; E-F, J-L, W-M, A-N-V, and C-G in upper case print; and a-o, b-f, and j-y in script.²⁰

Impairment of visual recognition. Most aphasic patients can match words to pictures to some extent, but many cannot point to the same words when spoken by the examiner, an example of the fact that severe reading deficits may result from impairment of auditory processes alone. However, patients with visual involvement tend to confuse words that look alike. *Match* may be read as *watch*, *dark* as *park*, and so on. Rate of word recognition is impaired. Patients frequently do not recognize words as units but must decipher each letter, then spell the word to themselves before they recognize it. They can sometimes be seen to trace letters in order to assist impaired visual recognition. Short words are recognized more easily than long words by such patients; they frequently decipher the first part of the word correctly and guess at the end of it. This is generally true of visual perception, but errors are exaggerated in aphasic patients with perceptual problems.

Impairment of visual recall. With impaired visual recall, one finds reversals and distortions of letter forms and substitutions of letters with similar visual configurations and of upper case for lower case letters, sometimes in the middle of a word. Spelling tends to be phonetic; these patients have particular difficulty with silent letters and double letters. Oral spelling usually exceeds written spelling; a word is often spelled aloud correctly and written erroneously. The

same errors appear day after day in all samples of the patient's writing, and identical errors are made by patient after patient.

Spatial disorientation. Spatial disorientation may or may not be associated with visual involvement. When it is present, patients have difficulty following the line and keeping the place both in reading and writing. Copying and drawing show marked distortions. The spokes in a wheel may be drawn horizontally, a house may be drawn with windows outside the walls, and a man may be drawn with eyes outside the head or one leg attached to the foot of the other at right angles. When spatial disorientation is gross, reading and writing are not apt to become functional skills.

In addition, patients frequently complain of blurring and obfuscation of vision, and perception varies markedly with conditions of illumination, size of stimulus, length of exposure, and background conditions.

If an aphasic patient shows the specific visual signs that have been described, the best inference would be a lesion in the posterior part of the language field; spatial disorientation would also be considered evidence of parietal involvement.

IMPAIRMENT OF MOTOR AND SENSORIMOTOR PROCESSES

Cranial nerve involvement. Some patients have observable paralysis or paresis of the speech musculature. With brain injury, vocal cord paralysis may be observed by laryngoscopic examination; unilateral or bilateral paralysis of the soft palate may be found; and certain movements of the tongue, usually protrusive and retractive but sometimes lateral, may be absent or weak, or the tongue tip may be elevated. All of these defects produce typical articulation deficits, characterized by distortion or substitution for sounds which require movements that the patient is unable to perform. These deficits are usually associated with difficulty in swallowing and are usually found with bilateral cerebral damage.

Sensorimotor involvement. Some aphasic patients have difficulty initiating and coordinating the movements of speech when no paralysis is present. These patients behave as though they did not know where the tongue was in the mouth or how to move it in a given direction or to a given position. Hughlings Jackson¹⁵ described a patient who put his fingers in his mouth to move his tongue in order to show the doctor that he knew what he wanted him to do and that he desired to cooperate. Evidence is accumulating which suggests that this kind of impairment

stems from reduction of proprioceptive and kinesthetic cues. Liberman,²¹ studying perception of phonemes at the Haskins Laboratory, has reported that, in the few cases where auditory and proprioceptive cues diverge, perception tends to follow proprioception. We have accumulated considerable data which show that, whereas patients with auditory impairment tend to confuse letters with names that sound alike and patients with visual impairment confuse letters that look alike, patients with sensorimotor impairment confuse sounds which require similar positions and movements for articulation, such as p-b-m, produced on the lips; t-d-n-l, made with the tongue tip and the upper tooth ridge; f-v, made with the upper teeth on the lower lip; and k-g, made with the back of the tongue and the velum.

Clinically, these patients make consistent articulation errors. The more complicated the motor pattern, the harder it is for them to pronounce a sound. They have more trouble with consonants with complex movement patterns than with simple ones, more difficulty with consonant blends and groups of consonants than with single consonants, and more trouble with polysyllabic than with monosyllabic words. All speech is laborious. Much-practiced words may be articulated smoothly, but a new word creates difficulty. Repetition at first tends to be very defective. They say such things as baco for bacon, with the final sound omitted; cosen for closet, with the l omitted and the n and t confused; and airper for airplane, with the r and l confused and the end of the word omitted. As speech emerges, patients with severe sensorimotor impairment tend to communicate largely in single words, but, as progress continues, phrases and sentences appear. The telegrammatic style of the early speech of these patients is characteristic of language in situations in which communication is difficult. In a study in progress at the Massachusetts Institute of Technology, linguistic analysis of the speech of such patients shows the same kind of curve as control tower data, which tend to support this hypothesis.

Aphasia, then, is a reduction of language in all modalities, upon which specific perceptual, motor, or sensorimotor difficulties may or may not be superimposed. Severity of involvement varies from patient to patient, and prognosis for recovery is more closely related to the pattern of involvement than to the initial severity level.

PATTERNS OF APHASIC IMPAIRMENT

Before classification of aphasic patients is discussed, it should be stated that a reliable test

cannot be obtained from an aphasic patient until he has become physiologically stabilized. No diagnosis can be made or prognosis stated during the acute phase of a cerebral injury, because it is impossible to know how much function will return spontaneously or what the residual deficit will be. Most authorities agree that all but a negligible percentage of spontaneous recovery occurs in the first three months after onset. Some patients stabilize more quickly than this; some, notably trauma cases, may take longer.

Treatment for aphasia requires the active participation of the patient. Motivation is rarely a problem, but physiologic status is. A patient who is not able to sit up for two hours without excessive fatigue or discomfort usually cannot be examined or treated successfully. He is not ready for examination or treatment until he is alert, noticing what is going on around him and beginning to respond to his environment. At this time, he should ordinarily be referred to a speech pathologist.

Aphasia requires long-term treatment — in most cases, years, not weeks or months. Treatment must also be intensive, meaning daily, at least at the beginning. In most situations, this is impractical, and the speech pathologist compromises by training a member of the family to participate in the necessary daily practice, carrying out instructions that change from week to week as the patient progresses.

To the present time, we have identified and studied 5 recurring patterns of aphasic impairment.^{22,23} There are probably other patterns which occur less frequently, but, conservatively, 95 per cent of our patients have fallen into 1 of these 5 groups.

Group 1. The first pattern is one of severe impairment in all language modalities. Two essential diagnostic criteria are (1) errors in pointing to common objects named by the examiner and (2) absence of functional speech, reading, or writing. Patients in this group cannot name objects, answer questions, or read or write spontaneously, although they can sometimes match simple words to pictures.

Prognosis for this group is poor. These patients rarely recover functional speech, though they can be taught to repeat, copy, and make associative responses. Some reactive speech appears but does not become voluntary. Auditory comprehension tends to improve with intensive stimulation, but regression takes place when stimulation is discontinued. The poor prognosis for this group is frequently hard to accept, for many group 1 patients are adequate in situations that do not depend on language. They

grasp some words and respond to total situations so well that families usually report that the patient understands everything which is said. They follow directions well if shown what to do and often show social awareness and appropriate social behavior. Treatment for aphasia is not indicated.

Group 2. Patients in this group show a general language deficit, with no specific perceptual or motor deficits. Diagnostic criteria are as follows: (1) no errors are made in pointing to common words named by the examiner, but verbal retention span is impaired; (2) some speech, reading, and writing abilities are usually preserved but are defective; (3) pronunciation may be exceedingly defective, but errors are inconsistent and can be corrected readily by ear; (4) some normal speech is usually present or emerges early; (5) vocabulary is reduced, and word-finding errors are common; (6) reading and writing reflect errors found in speech; and (7) errors increase in direct relation to the length of the stimulus or of the response required.

Prognosis for this group is excellent. Speech, reading, and writing improve as verbal retention span increases. Many group 2 patients have returned to exacting professions, such as teaching, medicine, or law. Students returning to classes usually find it necessary to take reduced course loads at first. Most universities have public speaking classes and reading clinics that offer effective help. Group 2 patients should have treatment; otherwise, residual difficulties persist, and they tend to settle for a lower performance level than is necessary.

Group 3. This group of patients are like those in group 2, but, in addition, have specific visual involvement. Diagnosis should not be made unless these signs are present: (1) distortion and reversal of letter forms; (2) confusion of symbols with similar visual configurations; (3) difficulty with silent letters; (4) a tendency toward phonetic spelling; (5) better performance on oral than written spelling; and (6) sometimes blurring and obfuscation of vision, difficulty following the line and keeping the place, and spatial disorientation.

Prognosis for recovery of speech for group 3 patients is the same as for group 2, but reading and writing improve more slowly. Functional skills are regained, but little inaccuracies tend to persist and rate of performance remains retarded. Group 3 patients cannot usually return to professions that place high demands upon speed and accuracy of reading and writing.

Group 4. Patients in this group show reduced

tion of language in all modalities which is usually severe, and, in addition, impairment of ability to imitate speech patterns without paralysis or paresis of the speech musculature. Such difficulties are considered cases of sensorimotor involvement. Articulation errors are consistent. The same sounds tend to be defective in all words, and sounds that require complex coordinations are more impaired than sounds with simple movement patterns. Many group 4 patients have such short auditory retention spans and such severely reduced vocabularies that they resemble patients in group 1 except that they do not make errors pointing to common objects named by the examiner.

These patients usually do not completely recover. Progress is slow, and language remains limited and sometimes defective, but it is functional as it is acquired. Prognosis depends more upon the amount of auditory impairment than on the severity of sensorimotor involvement. Group 4 patients require a long period of intensive retraining. Intensive auditory stimulation is necessary as well as facilitation of movements. Patients can usually achieve enough ability to communicate to be employable in situations in which language skills are not required.

Group 5. These patients show scattered auditory, visual, and motor findings. Cranial nerve involvement is usually present. The diagnostic criterion is involvement of all three of the basic processes involving language. Superficially, these patients differ from each other more than the patients in any other group, because the process most seriously involved varies with the individual. Some patients may show only slight impairment of auditory processes and only mild slurring of speech but have severe visual involve-

ment with spatial disorientation. In others, cranial nerve involvement is severe enough to almost preclude speech.

In age and etiology, group 5 is the most homogeneous of aphasic patients. Whereas in the other groups the entire age range and all etiologies were represented, in one sample studied here, all group 5 patients were over 50 years of age, and 75 per cent were over 60. In 8 per cent, the etiology was severe trauma; 8 per cent had advanced arteriosclerosis; and 83 per cent were known hypertensives who had incurred more than one known cerebral episode. Results are limited by the physiologic condition of the patient and the amount of generalized brain damage. Most patients in this group showed emotional lability, and few were capable of sustained or systematic effort. Most patients were more concerned about physical symptoms, such as dizziness, headaches, or dyspnea, than about recovery of speech. Some traumatic cases showed regressive behavior or evidence of organic psychosis. Although few group 5 patients could work independently, many did well when working with the clinician. Limited goals, such as increasing vocabulary or intelligibility, could be achieved. In many cases, this achievement contributed to the adjustment and well-being of the patient. Whether or not treatment is recommended should be determined by the patient's needs and his physiologic and mental status. Long-term treatment is seldom advisable.

Table 1 compares the mean number of errors obtained for each clinical group on the 6 sections of the Minnesota test, with the mean obtained for the total sample of 131 aphasic patients. Group means are expressed as a percentage of the mean for the total sample. While these

TABLE 1

MEAN NUMBER OF ERRORS FOR CLINICAL GROUPS ON SECTIONS OF THE MINNESOTA TEST FOR DIFFERENTIAL DIAGNOSIS OF APHASIA, EXPRESSED AS PERCENTAGES OF MEAN FOR TOTAL SAMPLE (N 131)

	A	B	C	D	E	F	G
Group 1	250%	180%	230%	210%	150%	280%	230%
Group 2	42%	42%	44%	33%	38%	23%	39%
Group 3	50%	50%	35%	70%	62%	50%	54%
Group 4	107%	80%	136%	93%	110%	61%	100%
Group 5	82%	110%	84%	110%	110%	105%	97%

- A. Tests for auditory comprehension
- B. Tests for visual and reading disturbances
- C. Tests for speech and language disturbances
- D. Tests for visnomotor and writing disturbances

- E. Tests for numerical concepts and arithmetic processes
- F. Tests for disturbances of body image
- G. Complete battery

figures differentiate the groups in the desired ways, the use of the diagnostic signs and criteria reported for the 5 groups does so more readily in the case of an individual patient.

On all sections except reading and numbers, group 1 averages more than twice the mean number of errors for the total sample on the complete battery. On section B, persons in group 1 are often able to pass matching tests. On E, they are frequently able to perform such tasks as making change and setting the hands of a clock to show what time they get up and go to bed, and sometimes they can solve simple numerical problems, such as adding 3 and 5, subtracting 7 from 13, and multiplying 3 times 5.

Patients in group 2 average less than half the mean number of errors for the total sample on all sections of the test. Group 3 is close to group 2 except on the writing and arithmetic tests, where, because of additional visual involvement, group 3 averages more errors. Patients in group 4 approximate the mean for the total sample, doing a little better on the reading tests, where the visual stimulus helps them, and significantly poorer on the speech and language tests because of sensorimotor involvement. In contrast, group 5 patients do best in comprehension and speech and have more trouble with reading, writing, and numbers, probably because cranial nerve

involvement must be extremely severe to make speech unintelligible, while visual involvement on a cerebral level affects reading and writing performances more severely. On the other hand, there is evidence that proprioceptive cues are important even in auditory perception of speech sounds,²¹ which may explain the poorer performance of group 4 on comprehension. Another possibility is that the combined severe auditory and sensory deficit may be related to lesions involving both auditory and secondary sensory-somatic areas.

Prognoses for the clinical groups—no significant recovery for group 1, excellent recovery for group 2, good recovery of language but slower progress in reading and writing for group 3, limited but functional gains for group 4, and recovery limited by physiologic and mental status for group 5—are remarkably reliable and have held up better than anticipated when applied to new samples of patients. The diagnostic task in aphasia is to assess first the general language deficit and then specific perceptual or motor disabilities that may or may not be present. Except for group 1 patients, perhaps, prognosis seems to be more intimately related to the latter findings than to the former, which is to say that the over-all pattern of impairment is more significant than the original severity level.

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Notes from a Medical Journey

Palinuro, Italy
12 May 1960

Dear Jay:

At Naples, 100 miles north as the crow flies, a mountain of records, blood, and food samples from Montegiorgio is being processed and Dr. Joseph Anderson, arrived from Minnesota for the purpose, is checking chemical methods with Dr. Flaminio Fidanza and his assistants -- Drs. Mancini, Cioffi, Vitale, Imbimbo, and Matteoli. As a result of conferences at Brussels a few weeks ago, we are preparing for some years of blood studies on European insurance applicants. La Prevoyance Sociale of Belgium will cooperate, and other companies in England, Sweden, and Germany may join the program already represented in the United States by the Lincoln National Life and the Mutual Service Insurance companies. We expect blood samples from about 5,000 men in the first year, and this figure could well be tripled. Naples will be the laboratory headquarters for this work, but the control will remain in Minnesota.

Unlike Dr. John Gofman's atherogenic index service in California, this is strictly a cooperative research program. By follow-up studies on life insurance applicants, we propose to examine the predictive value for health and disease of blood cholesterol as well as the characteristics ordinarily recorded for insurance purposes. The data will not be used concurrently in underwriting and we, of course, derive only scientific profit. Within three years, we shall have at least 25,000 men in the follow-up study, including men in the population surveys such as that just finished at Montegiorgio.

So far, follow-up studies in the United States indicate that a single blood sample analyzed for cholesterol allows one to identify a group of men whose risk of coronary heart disease in succeeding years is more than 3 times that of the rest of the men of the same age. This seems to be more important than the information from blood pressure and a great deal more significant than the relative body weight. But what is the risk up and down the cholesterol scale and how is this variable related to other char-

acteristics? Follow-up studies on a much larger scale are essential, and that is where the insurance companies can contribute uniquely.

Our beginning with a few insurance companies has been made possible by the method we have developed for the estimation of the serum cholesterol concentration from a few drops of finger tip blood allowed to dry on a bit of paper and analyzed at leisure at a distant laboratory. The cholesterol in this dried form is stable for many months and is readily extracted and measured, as we discovered years ago. The "gimmick," however, is to use whole blood without any measure of the amount at the time it is drawn. Fortunately, the concentrations of cholesterol in the red cells and of the sodium in the plasma are remarkably constant, and the balance of these substances between cells and plasma is such that the ratio of cholesterol to sodium in whole blood allows a very satisfactory estimate, for our purposes, of the cholesterol concentration in the serum. The ratio of cholesterol to whole blood solids is almost as good in all but persons with major blood disorders.

So now for a couple of days here in the sun at Palinuro, Ernest Klepetar and I are discussing all these matters and making plans for the future. Ernest, who steals time from his responsibility as actuary and vice-president of the Mutual Service Insurance Companies to aid in our epidemiologic research, has played a major role in securing the cooperation of the insurance companies, and the efforts of his president, Felix Rondeau, stimulated the meeting at Brussels. Ernest and his wife, Edith, are with us and, as we both have new Kharmann-Ghia sport coupes, identical save in color, we create a minor sensation in the little villages where such cars are novelties. At the moment a crowd of men is examining our cars down in the street below this little hotel (officially "third category" but very comfortable).

Everyone in Italy has heard of Palinuro because it bears the name of Palinurus, the faithful helmsman of Aeneas who, according to Virgil, was lost overboard off this cliff-bound shore, a victim of the God of Sleep, who drugged him with the waters of Lethe. Later, when Aeneas visited the Underworld from Lake Avernus, a few miles north of Naples, he met the shade of Palinurus, who bewailed his unburied state. The prophetic Sibyl, Aeneas' guide in the Underworld, consoled him with the promise that a tomb would be erected for him, "and that place shall bear the name of Palinurus forever."

And so it is, except that Palinurus' tomb is not to be found unless, most fitting, it be the lighthouse on the jutting headland which controls the sea from here to Capri, 60 miles distant. It was a delightful walk winding up the sheep tracks above the little fishing harbor, wild flowers in profusion everywhere, to the lighthouse, blinding white in the spring sunshine and much bigger than it seemed from the village a couple of miles away. As we looked up, wondering whether photographs were allowed, we were hailed from a high window and invited to come in and inspect. Two families tend the light, and we saw all the workings, set the light itself in motion, and drank in the blue of the Tyrrhenian Sea. Supplies come by

mule along the crest of the ridge, and the older man said it is a tranquil life. "Perhaps too tranquil," remarked the younger man, as he turned to gaze at the mountain behind us.

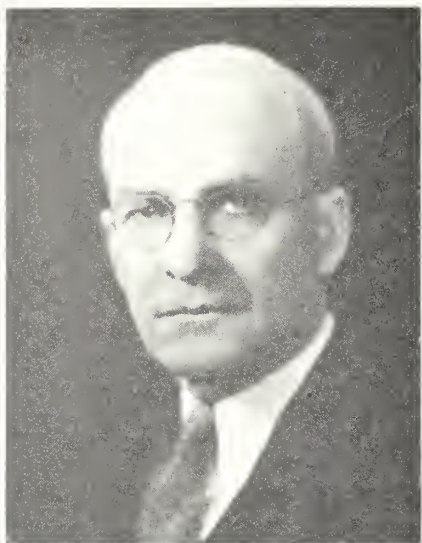
Later, sitting on the terrace of our hotel in the evening glow, fragments of conversation from the road and from balcony to balcony were broken by dogs barking. A donkey brayed, and then we noticed that the lighthouse had begun its nightly vigil -- short and long flashes in unvarying rhythm -- and we pictured the slow descent of the timing weight as we had seen it in the morning. Somehow it called to mind the lonely Greek temples at Paestum, halfway between here and Naples, reminder of the city that flourished there long before Rome and vanished, perhaps because of the constant plague of malaria on those flat lands by the sea. That medical problem continued until a few decades ago but is now practically forgotten, and the new settlers in those reclaimed areas are worried about economics and politics rather than malaria. For that matter, they also seem to escape two of our modern plagues, dental caries and coronary heart disease, though they have at least their share of peptic ulcer, liver disorders, and valvular heart disease.

But now the hotel owner is coming to discuss the important matter of what we shall eat for dinner and it's time to write, "Best wishes to all."

As ever,

A handwritten signature in dark ink, reading "Ancel Keys". The signature is fluid and cursive, with the first name "Ancel" written in a larger, more prominent script than the last name "Keys".

AK:ml



William Wallace Will, M.D.

J. ARTHUR MYERS, M.D.

Minneapolis

WHEN THEY REFER to Dr. William Wallace Will, Bertha, Minnesota, as being "always in a hurry," he willingly admits the claim except, of course, when he is caring for a patient. It could possibly be explained, for, in winter at least, he has to keep moving and fast, since, no matter how cold it is, Dr. Will refuses to wear a coat. They tell in Bertha that "Doc Will" has never had a cold and has never been ill.

He has stopped on several occasions, at least long enough to be paid tribute for his fifty-four years as a practicing physician in the town of Bertha. Dr. Will was nominated as the Best Family Doctor in the nation in 1948, with the backing of the entire Minnesota State Medical Association. In his selection as the outstanding general practitioner in Minnesota, he represented the state's more than 1,500 general practitioners in a national contest sponsored by the American Medical Association.

What the town of Bertha, its population of 550, thought about the honor can best be described from an excerpt from *The Bertha Herald*, the local newspaper, in the January 8, 1948, issue which read: "When Doctor Will's Minnesota colleagues offered his name as Minnesota's best, they simply acknowledged what we in Bertha have accepted as a fact right along. We are continually amazed at the tribute paid him and at the devotion he has earned in his years of service. It is richly deserved for he had never spared himself where a patient was concerned. He has worked hours, days, and even weeks with little rest or sleep when there has been a need for his considerate care. All of this has been given freely without regard to the patient's financial standing."

Dr. Will is one native son who preferred to remain in his home state rather than try out greener fields, which, to a man of his superior ability, must have beckoned him many times. He was born in Blue Earth County, Minnesota, on May 14, 1879. His parents were natives of Scotland. His father came to this country from Dundee in 1856 and his mother

from Perthshire, Scotland, in 1844. The Scotch ancestry stood young Will well indeed, as he was to tax heavily his physical endurance and even his disposition over the many years he has faithfully served as physician and surgeon and health mentor for the Bertha community. The hardships of those early pioneer days were well instilled, preparing him for his long experience in service to others. The Will family was a large one of 10 boys and 1 girl.

Will entered the University of Minnesota following graduation from Mapleton High School and, in 1905, was awarded the degree of Doctor of Medicine. He set up practice in Bertha immediately following his internship in 1906. It has been in Bertha and the surrounding community that Dr. Will has carried on his practice as physician and surgeon for a half century with such fine leadership that state and national honors have been brought to his door. As for Dr. Will, the high honors accorded him are much appreciated but are as nothing to him compared to the love and appreciation which come to him from his "home folks." Soon after he came to Bertha to begin practice, he married, in 1907, a Mapleton girl, Miss Marianna Bishop. In the Will family, 2 of the brothers married sisters. Dr. Will's wife and her sister, Miss Kathryn Bishop, were daughters of Dr. and Mrs. Ira Bishop of Mapleton. Two years after Dr. and Mrs. Will were married, Dr. Melville Will married the second Bishop girl. They, too, moved to Bertha, and the couples continued together until parted by death. Dr. Will is the only survivor.

The country doctor's wife was also deserving of an award as the "perfect doctor's wife," for over the years she gave loyalty, assistance, and unselfish devotion that only a doctor's wife understands. At his side, she became, as he did, a part of the community life of Bertha, participating actively in church, social, musical, and educational circles. The couple reared 4 children.

Dr. Will was forced to labor under the many untold

difficulties that were present or sprang up before those early practitioners. But he took the hurdles without complaint and in the same stride as that of the Scot who said, "if at first you don't succeed, try, try, again." A friend and colleague of many years, Dr. E. J. Simons, Swanville, Minnesota, who, with Dr. H. E. Hilleboe, conducted the first extensive tuberculosis survey by private practitioners in Minnesota in 1929 and 1930, has often lauded Dr. Will. Of him, Dr. Simons, president of the Minnesota State Medical Association in 1946, said at a gathering arranged to honor the Bertha practitioner, "the immense strides made in the practice of medicine were through the efforts of men like him."

A great interest in the study of tuberculosis and untiring work among his patients who had contracted the disease was demonstrated by Dr. Will all through his professional career. He is a veteran of sanatorium commissions and was listed in 1949 as 1 of the 4 physicians serving the longest terms. He had represented Fair Oaks Lodge Sanatorium, Wadena, Minnesota, which at that time had served Wadena and Todd counties for thirty-one years.

In the early days of his practice in Bertha, Dr. Will cared for the sick at his office and at patients' homes. It was some time before a 20-bed hospital was erected in Bertha. Now his offices are housed in an 8-room brick clinic built about 1941. Sharing the offices with him were his brother, the late Dr. Melville B. Will. Melville, given his mother's maiden name, was born in Mapleton on February 2, 1882. It is significant that it was in the same year Melville was born that the world heard of one of the greatest discoveries in the field of medicine, the isolation of the tubercle bacillus by Robert Koch. Dr. Melville received his early education in Mapleton, and, after high school, he, too, entered the University of Minnesota. However, his choice was in another field, and he received his degree from the College of Dentistry in 1908. He began practicing dentistry in Bertha two years after his brother settled there. The same year he married Miss Kathryn Bishop. They had 4 children, 1 of whom, Dr. Charles B. Will, worked for sometime at the Thiel Hospital in Bertha. Of his uncle, Dr. Charles B. Will says "he was a typical horse and buggy doctor. He had 6 to 8 horses which he drove hard all fall and winter in making calls. Several times, when roads were too drifted, he actually made calls on horseback. He seemed to thrive on it. He still bounces or runs upstairs at the hospital."

A testimonial to the skill of Dr. Will are the throngs of patients that daily visit the clinic with "standing room only." They are not only from Bertha and vicinity but from far distant places.

Honors have come from afar to Minnesota's noted son, now a famed physician. In 1945, he was the only practicing physician in Minnesota serving in a rural area to have been granted fellowship in the American College of Surgeons. For another high honor, Dr. and Mrs. Will went to Washington, D. C., in 1945, where he was elected fellow in the Interna-

tional College of Surgeons at the national convention of the organization. This honor and recognition of Dr. Will was one given to few men engaged at the present time in the practice of surgery. The membership of this organization is limited to surgeons acknowledged by the medical profession as leaders, pooling their knowledge and the results of their research, thus giving the benefit of their studies to medical men throughout the world.

Dr. Will is widely known for his willingness to share his experiences with others and to encourage young men in the profession. He is a charter member of Xi chapter of the University of Minnesota's Phi Beta Pi medical fraternity and attends many functions of the group. His nephew, Dr. Charles B. Will, is also a member of Xi chapter. Dr. Will keeps up with the latest developments in medicine, and all through his career he has maintained an ardent desire to continually improve his skills. He was president of the Minnesota State Medical Association in 1939, and other medical associations in which he holds memberships include the Todd County Medical Society, the Upper Mississippi Medical Society, and the American Medical Association. He grew up with Bertha and has been one of the keenest "boosters" for his town. He once served as president for the local Commercial Club.

Bertha went to town in a big way for him on the occasion of the fortieth anniversary of the establishment of his practice in the village. The townspeople, augmented by many from neighboring Mapleton and the surrounding area, proclaimed Sunday, September 22, 1946, as "Doctor Will Day." In the Bertha high school auditorium where the festivities took place, the largest crowd ever to assemble under one roof in the history of the village made the walls bulge with happy people. The 2,000 who gained entrance were lucky, and another 1,000 waited in halls and corridors and outside for the chance to congratulate their "Doc Will" and his wife.

The celebration was featured by a parade of the 4,000 babies whom Dr. Will had helped to deliver during his forty years of practice. The first baby he delivered, Arthur Steinberg of St. James, Minnesota, now a strapping man the size of the doctor, was there to pay his respects, and the latest baby he had delivered at the time, 5-day-old Steven Lee, had his picture taken with Dr. Will.

Representing the people of Minnesota, the current Governor Edward J. Thye extended greetings and congratulations to Dr. and Mrs. Will. He spoke on Dr. Will's widespread reputation as an outstanding doctor of the state and of how he had heard of him many years before he had the pleasure of meeting him. "This is one of the nicest groups of people I have ever seen gathered together," he said, "and I consider it one of the highest compliments I have ever been paid to have been asked to address this meeting." Dr. Will responded by saying that the display of friendship tendered to him was one of the nicest memories of his forty years in Bertha. He

climaxed his remarks by saying that he felt that in establishing his practice in Bertha forty years ago, he had chosen well and that his greatest desire was to spend the rest of his life in this community, being of service to his friends.

Dr. Will, now in his half century of service to his community, is in as much of a hurry as ever, continuing his practice in the service of his town and the adjoining areas. A telegram sent for the fortieth anniversary celebration from the mayor of Sauk Center, Minnesota, read: "There is a reason that folks say, where there's a Will there is a way." And Dr. Will's way is dedicated service.

At the fiftieth anniversary of his practice in medicine in 1956, he received his 50-year button, the

special award of the Minnesota State Medical Society.

Each person has but one life. The service which one renders must always be given first place. It will always be true, as it has been in the past, that those who place service rendered first and treat their fellow men honestly and squarely and give the very best that they have been able to obtain in a most conscientious manner will not only retain their own self-respect but will command the respect of all with whom they come in contact. This and much more has been demonstrated by Dr. Will in all of his life and work.

The author wishes to thank Miss Dorothy Riley for her assistance in the preparation of this manuscript.

THE TENDENCY of intravascular erythrocytes to aggregate and cause slowing of microcirculation becomes greater as the degree of diabetic retinopathy and nephropathy increases. Changes in distribution of serum proteins, lipoproteins, and protein-bound carbohydrates are associated with vascular degeneration in diabetic patients. The relationship between such degeneration and the high incidence of erythrocyte aggregation observed biomicroscopically *in vivo*, therefore, reflects changes in blood protein distribution. Aggregation seems to be related more closely to an increase in α_2 globulin and decrease in albumin content than to increases occurring in other protein fractions or conjugates. Vascular pattern changes and erythrocyte clumping apparently are independent manifestations of tissue reactions to metabolic alterations of diabetes.

J. DITZEL and P. MOINAT: Changes in serum proteins, lipoproteins, and protein-bound carbohydrates in relation to pathologic alterations in the microcirculation of diabetic subjects. *J. Lab. & Clin. Med.* 54:843-859, 1959.

INDIVIDUALS intermittently exposed to small amounts of carbon monoxide fumes may have persistent symptoms and signs, particularly of an organic confusion state, between exposures. The manifestations are exacerbated by exposure and correlated with high, fluctuating blood levels of carbon monoxide.

The most common symptoms are anorexia, nausea, weight loss, apathy, fatigability, headache, dizziness, insomnia, and personality disturbances. Neurologic signs may include ataxic gait, hyperreflexia, nystagmus, tremor, general weakness, alterations in perception of pain, dysdiadochokinesia, myoclonic twitching, hemiplegia, aphasia, anisocoria, anosmia, and palsy of the facial and glossopharyngeal nerves. Electroencephalographic examination reveals diffuse and local epileptiform discharges that disappear gradually. Electrocardiographic abnormalities, particularly extrasystoles and ST changes, are occasionally observed.

Differential diagnosis must exclude epilepsy, organic brain syndromes, and hyperthyroidism.

G. J. GILBERT and G. H. GLASER: Neurologic manifestations of chronic carbon monoxide poisoning. *New England J. Med.* 261:1217-1220, 1959.

a
three-year
case
history²

Date	Entries and Comments	Orinase Dosage (grams per da		
		1	2	3
6/21/56	Mr. J. S., 54 yrs. old, diabetes mellitus 1½ yrs. F. H.—neg. for diabetes. P. H.—surg. 0, med. 0. Restaurant manager—M., 3 children living and well. Wt. 155 lb. 1 yr. ago; now 125. P. I.—onset 1½ yrs. ago with thirst and polyuria; glycosuria found. On diet. In past year lost 30 lb., strict diet—all kinds of dietetic substitutes. Some asthenia. Afraid of insulin. No recent glycosuria or nocturia. P. Exam.—thin male. Fundi neg. ENT neg. BP 140/80. Heart and lungs neg. Extremities: poor pulses. Urine—sugar 0, acetone 0. Noon blood sugar 240. Rx more adequate diet and Orinase 3 Gm.			
7/9/56	Wt. 127, urine 0-0. B. S. 110, occ. nocturia but no glycosuria. Rx: eat more, Orinase 2 Gm.			
8/29/56	Wt. 139½, urine 0-0-0. B. S. 205. Rx 1.5 Gm.			
10/1/56	Wt. 143, urine 0-0-0. B. S. 125. Rx 1 Gm.			
11/5/56	Wt. 148, urine 4+ -0-0, noon B. S. 160. Rx 2 Gm.			
12/7/56	Wt. 146, urine 0-0-0, noon B. S. 120. Rx 1 Gm.			
1/11/57	Wt. 144½, urine 0-0-0, noon B. S. 150. Rx 1 Gm.			
3/11/57	Wt. 144½, urine 0-0, noon B. S. 120. Rx 1 Gm.			
5/10/57	Wt. 140½, urine 4+ -0-0. B. S. 275. Rx 3 Gm.			
6/12/57	Wt. 138½, urine 0-0-0, noon B. S. 114. Rx 2 Gm.			
8/7/57	Wt. 136½, urine 0-0-0. B. S. 100. Rx 1 Gm.			
10/2/57	Wt. 136½, urine 0-0-0. B. S. 85. Rx 1 Gm.			
11/29/57	Wt. 136½, urine 0-0-0. B. S. 123. Rx 1 Gm.			
1/17/58	Wt. 134¾, urine 4+ -0-0. B. S. 216. Rx 3 Gm.			
2/14/58	Wt. 132, urine 0-0-0. B. S. 135. Rx 3 Gm.			
3/28/58	Wt. 136½, urine 0-0-0, noon B. S. 93. Rx 2 Gm.			
5/5/58	Wt. 138, urine 0-0-0. B. S. 112. Rx 1.5 Gm.			
6/16/58	Wt. 137, urine 0-0-0. B. S. 93. Rx 1 Gm.			
8/11/58	Wt. 138, urine 0-0-0, noon B. S. 132. Rx 0.5 Gm.			
10/20/58	Wt. 134, urine 3+ -0-0. B. S. 220. Rx 1 Gm.			
11/24/58	Wt. 131½, urine trace -0-0. B. S. 251. Rx 3 Gm.			
2/18/59	Wt. 134, urine 0-0-0. B. S. 120. Rx 1 Gm.			
4/9/59	Wt. 137½, urine 0-0-0. B. S. normal. Rx 1 Gm.			
6/5/59	Wt. 138, urine 0-0-0. B. S. normal. Rx 1 Gm.			

BOOK REVIEWS

Manual of Skin Diseases

GORDON C. SAUER, M.D., 1959. Philadelphia: J. B. Lippincott Co. 269 pages. Illustrated. \$9.75.

This book was motivated by a senior medical student who asked: "Where can I find a good 50-page book on dermatology?" Exclusive of pictures, this comes close to the perfect answer. It is highly recommended to any student or practitioner who wants to learn basic dermatology. It is reminiscent of an improved and expanded version of the World War II *Armed Forces Manual of Dermatology*. Its "much in little" has not been rivaled since that handbook.

There are 28 pages of color plates. Costs of these plates were defrayed by various pharmaceutical companies—a form of advertising that should be commended. Both the colored and the 151 black and white pictures are outstanding for teaching value and for reproduction. I usually look at an illustration of a given dermatologic entity and, upon reading the label, agree that the picture might represent what it is alleged to portray but feel that it might also be 6 other things. A dermatologist should be able to diagnose correctly over 90 per cent of the pictures shown in this text.

One thing not seen in previous textbooks of dermatology is the expansion of a given area from a drawn diagram to a blowup of the area, shown as a photograph adjacent to the diagram and fastened to it by expanding lines. The same diagrams, for example, atopic eczema, pityriasis rosea, and secondary syphilis, are used throughout—a highly effective teaching method.

This text is written from the viewpoint of a complete stranger to dermatology. Since the author is assistant clinical professor of dermatology and chief of the Section of Dermatology at the University of Kansas Medical School, it is amazing that he still has the rapport and empathy to be capable of writing such a bridge—a pons asinorum between total ignorance and his own erudition.

Outstanding and unusual in the 27 chapters are chapters IV, XXV, and XXVII. In chapter IV, "Your Introduction to the Patient," a conversational example of how to take a dermatologic history is given. The silly answers given by the patient will certainly ring true to anyone who has done this. Chapter XXV, on Geographic Skin Diseases in North America, should be useful to almost anyone. Chapter XXVII, on "Basic Dermatologic Equipment," is a gem that should be given as a reprint to students as they finish board examinations. It would save a practitioner who wanted to buy dermatologic equipment 10 times the price of the book and is only 3 pages long! Inexpensive apparatus that do as well or better than much more expensive equipment offered are strikingly pointed out.

The book is written in almost conversational style. It is exceptionally easy to read, interesting, and anecdotal throughout. The anecdotes are not dragged in but are germane, pertinent, and illustrative of some instructive point. The language avoids polysyllables. Words are short

and of Anglo-Saxon origin. All discussion is blunt, terse, pertinent, and intensely practical. Examples given are so striking and colorful that they should be easily retained, even by students or practitioners with jaded memories. There are many "pearls"—in some places, the author forthrightly labels the collections "pearls." Therapy is forthrightly labeled good, bad, or indifferent. When therapy is poor, as for hirsutism in women, it is listed first in terms of what is least difficult, least expensive, least dangerous, least damaging and so forth, with progressive steps toward more effective but more difficult, dangerous, or expensive procedures.

The diplomate in dermatology will probably not learn much from this book and is not intended to. However, even he might buy it as a model of how to explain skin disease to patients in terms that they will understand. This book will be read by most purchasers like a novel. It will allow the general practitioner to be a better "do-it-yourself" dermatologist. While study of any material would encourage this, this text is less likely to allow a practitioner to get into trouble.

This book should be a medical best seller. At \$9.75, it is the best bargain available since the passing of the nickel cup of coffee.

MURRAY C. ZIMMERMAN, M.D.
Whittier, California

Metabolic Aspects of Renal Function

WILLIAM D. LOISPEICH, M.D., 1959. Springfield, Ill.: Charles C Thomas. 190 pages. Illustrated. \$7.50.

The author has brought together 7 chapters—an introduction and 6 essays—which have a common renal metabolic basis. This volume is not intended as a textbook of renal physiology but is really a compilation of nicely related topics on which the author has done research for many years. In this sense, it is very personal and makes for splendid reading. The material is neither exhaustive nor exhausting but uses as a frame of reference important contributions made by others as well as the author.

Each chapter except one is cleanly rounded with a summary and a table of references. The text is well illustrated with 58 informative figures. The literary style is simple and direct, which makes the reading of this difficult and technical material a pleasure rather than a chore. In addition to the very informative introduction are chapters dealing with the transport and metabolism of phosphate, amino acids and glucose, the tricarboxylic acid cycle in the kidney, the synthesis and secretion of ammonia, organic acid and bases, and phorbizin.

The only very minor criticism is the too frequent use of the word "beautiful" to describe a brilliant experiment. This book is worthwhile reading for all who share the author's interest in the metabolic aspects of renal function.

ALBERT J. PAQUIN, JR., M.D.
New York City

(Continued on page 32A)

CLINICAL REMISSION IN A "PROBLEM" ARTHRITIC

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BOOK REVIEWS

(Continued from page 30A)

Intussusception in Infants and Children

MARK M. RAVITCH, M.D., 1959. Springfield, Ill.: Charles C Thomas. 119 pages. Illustrated. \$9.00.

Dr. Ravitch has long been an advocate of reduction of intussusception by barium enema. This well-compiled and beautifully illustrated monograph serves to defend his position.

The book begins with a complete review of the history of the subject, and the assembled literature is referred to frequently in the succeeding sections.

The clinical picture of this disease is described fully, along with the incidence related to age, race, season of year, and so forth of 199 cases of intussusception seen at the Johns Hopkins Hospital from 1893 to 1958. These facts are well illustrated by charts and are correlated with the reports of others.

The chapter on "Diagnostic Errors" is very worthwhile. The important errors are reviewed and accompanied by brief, illustrative case reports and clear reproductions of roentgenograms.

Another section is devoted to experimental studies that are well documented and beautifully illustrated. The main purpose of this work is to prove, by barium enema and pathologic examination, that reduction of experimentally produced intussusception by hydrostatic pressure is without danger if properly done.

The remainder of the book relates the various operative and nonoperative methods of treating this condition. Techniques of coping with irreducible intussusceptions are described, and the merits of each are discussed. Presented are some unusual serial roentgenograms of reduction of intussusceptions by barium enema. The ease for this method of treatment is well presented, and its limitations are described.

The book is well bound and printed on high-grade paper. The illustrations in color and black and white are excellent. The text is clearly, concisely, and objectively written.

This volume would seem to be of interest to anyone doing pediatric surgery. Pediatricians and general surgeons might also benefit from its contents, and the intern and resident will find in it a complete coverage of the subject.

WILLIAM L. RIKER, M.D.
Chicago

Survey of Clinical Pediatrics

LAWRENCE B. SLOBODY, M.D., 1959. New York: McGraw-Hill Book Co., Inc. 530 pages. \$11.00.

This book was designed primarily for the student and general practitioner, "focusing on the high lights and relationships of pediatrics." The author succeeds in these goals admirably. The book is current and fulfills a classic need for an easy-to-read, comprehensive, exam-preparing text. Each chapter ends with pertinent examination type of questions, which cover the topics well. Unfortunately, the author uses "majority opinion" on controversial subjects without the help of any quoted references. This lends to a certain obvious rigidity in the subject material, thereby making this work quite limited. Constant revisions of the text can be foreseen as "majority opinion" alters. Within these narrow limits, as a current review of pediatrics, this book is recommended, though more highly for students than for practitioners.

HERSCHEL J. KAUFMAN, M.D.
Minneapolis

The Journal Lancet

SERVING THE MEDICAL PROFESSION OF MINNESOTA,
NORTH DAKOTA, SOUTH DAKOTA, AND MONTANA

Diabetic Emergencies

HOWARD F. ROOT, M.D.,
and ODETTE VEIT, M.D.

Boston

ANY EMERGENCY—medical or surgical—may occur in a diabetic patient. Such an emergency may acquire unusual features which are dependent upon the patient's diabetic status. Present concepts of the pathologic physiology and natural history of diabetes must be kept in mind when approaching the problems frequently presented by surgical or other emergencies. Diabetes mellitus is a hereditary and inborn error of metabolism. In its clinical form it is a complex state characterized by both catabolic and anabolic abnormalities in carbohydrate, fat, protein, phosphate, and potassium metabolism in many tissues; by pathologic changes in acid-base balance and in the synthesis of vital cofactors; by changes in enzyme activity; and probably by alterations in cell permeability. Genetic factors may play an important part not only in determining the time of onset of diabetes and the pre-diabetic state but also in determining the susceptibility of the individual to premature vascular disease. Genetic factors perhaps also determine an individual's innate resistance to infection.

GENERAL CONSIDERATIONS

The mild diabetic, or the patient with diabetes of relatively short duration, who is treated early

and continuously with proper diet and insulin, may present no great problems in addition to those problems inherent in the emergency itself. On the other hand, diabetic patients in whom unrecognized diabetes has been present for years, or in whom treatment has been inadequate and the use of insulin has been long delayed, may present metabolic problems which gravely affect: (1) diagnosis, (2) the course of treatment, and (3) the ultimate prognosis.

Even in the supposedly mild diabetic patient, a period of stress may be followed by a sharp decline in carbohydrate tolerance; this decline results from increased activity of the sympathetic nervous system. The influence of epinephrine and a rise in glucocorticoid secretion by the adrenal cortex may be accompanied by increased glucose discharge from the liver or by increased carbohydrate formation from protein and fat. When infection is added to the situation, these combined elements may gravely affect even the mild case of diabetes. In patients with unrecognized and uncontrolled diabetes, various nutritional and metabolic disturbances, as well as ketosis, may combine to complicate an already difficult situation. States of diminished consciousness, resulting from hypoglycemia or ketosis, may contribute to accidents of various types.

Problems resulting from traffic accidents or other mishaps may become greatly magnified in accordance with the particular state of the patient at the time. Thus, in diabetic patients who are already hypoglycemic, trauma with resulting concussion of the brain may have the more

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serious consequences of: (1) failure to recognize the hypoglycemia in the first place and (2) serious damage to the brain with consequent difficulty in treatment and prolonged recovery.

If the patient is in an early stage of acidosis, or if diabetes is uncontrolled, defects in vision may play an important part in accidents. That such accidents are preventable has been indicated by a study of diabetic patients at the Joslin Clinic, Boston. In a large series of such patients, it appeared that those who had received careful instruction in hospital teaching classes had actually had fewer automobile accidents than their marital partners.

The diabetic patient whose diabetes has long been neglected or inadequately controlled without skillful use of insulin and proper diet may present not only acute problems of ketoacidosis, but also those problems of nutritional deficiency resulting from loss of weight, protein deficiency, and disturbed acid-base balance. In a group of diabetic patients at the New England Deaconess Hospital, Boston, the loss of weight preceding such procedures as transmetatarsal amputation varied from 34 to 120 lb. In the severe diabetic, the formation of carbohydrate from protein by deamination may be excessive. Since nutritional anemia may accompany protein deficiency, the need for restoring protein deficit may be of extreme importance for successful convalescence. Because such patients often suffer from osteoporosis and other disorders, the healing of fractures may present difficulties.

The most difficult problems occur in the patient with basically severe diabetes, with onset in early life, who presents the typical renal complication — diabetic nephropathy (Kimmelstiel-Wilson syndrome). A history of edema, albuminuria, visual disturbance, and the finding of hypertension and albuminuria, with or without elevations of blood urea nitrogen of the serum, may clearly indicate an advanced stage of the condition. This condition may have begun years before as premature vascular disease involving arterioles, capillaries, and the larger vessels. Here limitations are placed upon protein feeding; problems in electrolyte balance are common, and if nitrogen retention is present, dangers may exist not only in the use of antibiotics but of simpler drugs. If glomerular filtration is impaired, retention of drugs or antibiotics may occur with serious toxic effects. In such cases, the nephrotoxic effects of streptomycin, neomycin, and bacitracin may be greatly intensified. Even much simpler drugs, such as aspirin, have been known to have serious toxic effects in such patients. It must be remembered that far-advanced

pyelonephritis, with or without the other features of diabetic nephropathy, may exist without present or past symptoms being known to the patient.

Antibiotic-induced diseases include a number of serious conditions which are dependent upon the destruction of susceptible organisms, thereby permitting overgrowth of resistant organisms. In patients with diabetes of long duration, the use of antibiotics is therefore not a matter of routine. Such use is dependent upon careful consideration of the individual; it usually requires access to a laboratory where specific tests can clearly demonstrate the susceptibility of the organism, found by culture, to various antibiotics.

The use of oral hypoglycemic agents should not be begun at the time of surgery. Indeed, it is a common rule that during and after surgery, particularly in the presence of infection, insulin must always take the place of oral agents. It must be remembered that no oral agent is effective without insulin. The patient must either secrete sufficient insulin in his own pancreas or else receive insulin by injection. A properly planned diet is essential in all cases. Oral hypoglycemic agents administered without proper dietary control lead sooner or later to reactivation of the diabetes.

An almost infinite variety of emergency situations may be affected by the state of diabetes in a given patient. Only two emergencies characteristic of diabetes will be here considered—hypoglycemia and diabetic coma.

HYPOGLYCEMIA

In treating diabetes with insulin, the prevention of severe episodes of hypoglycemia is an important problem. Mild hypoglycemia is a common occurrence both in the hospital and in the ordinary life of a patient during treatment outside the hospital. Mild hypoglycemia is of little importance, since prompt treatment with food is usually carried out. It is probably true that a young patient with moderately severe diabetes cannot be kept under adequate control without occasional periods of hypoglycemia.

On the other hand, severe hypoglycemia may have serious consequences by affecting judgment or success in passing examinations and, occasionally, by causing serious accidents or social disasters. The fear of an insulin reaction may be exaggerated, resulting in excessive amounts of carbohydrate being taken. As a result of the bad test, the insulin dose is then steadily increased. The result is continued lack of diabetic control and the eventual development of severe vascular lesions in eyes, kidneys, and heart.

It is important that diabetic patients avoid

frequent and serious insulin reactions. Fortunately, almost all reactions are avoidable. One difficulty arises in diabetic patients who have been taking insulin for ten to fifteen years or longer. There comes a period when these patients no longer recognize the warning symptoms advising them of the importance of taking food—symptoms which had been thoroughly reliable and dependable for years previously. These patients are in the unstable group. Careful study of a series of more than 100 such patients reported by Balodimos and Root¹ disclosed no common factor. However, it did make possible the tentative hypothesis that some change in their central nervous systems might be responsible for the failure of these patients to promptly recognize symptoms which they had been easily recognizing for years. Nevertheless, the management of this group of patients is possible if more frequent feedings of carbohydrate, more careful measuring of the diet, and more careful use of insulin are maintained.

Fatalities are rare. However, apparently mild reactions may go rapidly to a severe stage. This makes it necessary to regard every episode of hypoglycemia, in a sense, as an emergency which requires that carbohydrate be taken immediately to bring the episode to an end.

Symptoms. When the well-known symptoms of sweating, nervousness, tremor, faintness, hunger, headache, rapid heart action, and double vision or unsteady gait develop in an individual within a few hours after taking insulin, he probably is suffering from hypoglycemia. Occasionally a patient may by accident go into unconsciousness and remain in that state for some time. Under conditions of long-continued hypoglycemia, serious damage may result. It is important that diabetic patients carry some identification to indicate not only that they are diabetic but also what dose and type of insulin they are taking.

Patients who receive unmodified or clear insulin may have a maximal fall in blood sugar, and therefore hypoglycemic symptoms, three or four hours after the dose. After insulin of long duration, such as globin or NPH, reactions may occur at a later interval, such as eight to ten hours. With such insulin as protamine zinc or ultra-Lente insulin, reactions may occur from twelve to thirty hours after the dose. Unusual exercise or an unusually meager diet, and particularly the omission of a meal or the occurrence of diarrhea, will make episodes of hypoglycemia more likely to occur.

The occurrence of an insulin reaction during high blood sugar levels is extremely rare, if it

does occur at all. However, if the blood sugar falls rapidly, sometimes symptoms do develop by the time the blood sugar has reached normal or nearly normal levels. Because the patient is unconscious or in severe hypoglycemic shock, neither he nor his family can explain the circumstances precipitating the reaction. Therefore, when the patient has a urine that shows much sugar, confusion in diagnosis often arises. This occurs particularly where the blood sugar fall has been rapid and the urine tested has been in the bladder for several hours. Therefore, patients are usually instructed to test a second voiding of urine when making a decision in regard to the next insulin dose. The instruction of patients in the use of insulin, diet, and exercise is of prime importance. It is still true that, when possible, all diabetic patients should begin their treatment with a period in a hospital which has a well-organized teaching unit. This teaching will not only prevent emergencies from hypoglycemia but will also prevent diabetic coma and lessen the frequency of other mishaps.

Almost any symptoms in the central nervous system may at times be due to hypoglycemia. A series of 5 characteristic stages has been described by Himwich² in patients in whom hypoglycemia is induced for the treatment of schizophrenia. In patients found unconscious, a striking feature is that the skin is moist. The pulse rate may be rapid and the blood pressure slightly elevated or normal. Twitching or convulsive movements may be present. Opisthotonus may occur.

Treatment. In an unconscious patient, the immediate administration of a glucose solution, preferably in 50 per cent concentration, is indicated. It is our rule to continuously administer the solution in patients known to have insulin hypoglycemia until such patients arouse. Sometimes 50 to 75 gm. of glucose has been necessary. It may be a fatal mistake to omit glucose administration after the first injection, hoping that recovery will take place, since in some cases irreversible damage to the brain may rapidly occur. Patients may remain unresponsive for days or even for weeks and still recover.

In table 1, the causes of hypoglycemia are summarized. In the diabetic patient, injected insulin is the most frequent cause. However, other factors (listed under "Spontaneous hypoglycemia") may occur in the diabetic patient and contribute to the hypoglycemic state. A wide variety of organic conditions are listed in table 1. The functional causes (listed under "Excess of insulin" and "Loss of glucose") are rarely of importance in true diabetic patients.

TABLE I
CAUSES OF HYPOGLYCEMIA

1. Excess of injected insulin
 - a. Diabetes mellitus
 - b. Factitious hyperinsulinism (surreptitious administration)
2. Spontaneous hypoglycemia
 - a. Organic (recognizable anatomic lesion)
 - Increased insulin:
 1. Spontaneous hyperinsulinism
 - Pancreatic islet adenoma: simple, multiple, and aberrant
 - Pancreatic islet cell carcinoma: localized or with metastases
 2. Central nervous system lesions: hypothalamus or brain stem
 3. Hyperplasia of the islets of Langerhans
 - Deficiency of insulin antagonists:
 1. Anterior pituitary hypofunction: destructive lesions (tumors and cysts), atrophy, and degeneration
 2. Adrenal cortical hypofunction
 - Deficiency of available glucose:
 1. Hepatic disease (choleangiolitis, toxic hepatitis, fatty degeneration, von Gierke's disease)
 - b. Functional (no recognizable anatomic lesion)
 - Excess of insulin:
 1. Hyperinsulinism (imbalance of the autonomic nervous system): functional hypoglycemia, reactive hypoglycemia
 2. Alimentary hyperinsulinism (rapid intestinal absorption) After gastroenterostomy, after gastric resection (partial or total)
 - Loss of glucose:
 1. Renal glycosuria (severe) lactation, severe inanition, severe continuous exercise

DIABETIC COMA

Ketosis and coma remain the most characteristic emergencies of diabetes. The word "coma" suggests profound unconsciousness, but the patient in whom diabetic ketosis is developing may be in danger long before he is unconscious. Therefore, the use of the words "diabetic coma" for early as well as late stages is justified. For a summary of definition, etiologic factors, pathophysiology, and factors in electrolyte balance in diabetic ketosis, see the appendix.

Wide variations in severity of diabetic ketosis and acidosis occur, and various standards of definition have been employed. In order to compare results in one period with those in another, it has been customary for many years at the Joslin Clinic to classify any case of diabetic acidosis or ketosis as one of coma when the carbon dioxide combining power, or more recently the carbon dioxide content, of the blood plasma is 20 vol. per cent (9.0 mEq./l.) or less. Actually, this dividing line was accepted shortly after the introduction of insulin. The clinical grounds were that, before the use of insulin, very few patients with diabetic ketosis recovered if the CO₂ content of the blood was below this accepted figure. Deaths from diabetic coma have

become less frequent, but they still do occur because of ignorance, delay in diagnosis, or other factors. Actually, the percentage of admissions in diabetic coma at the New England Deaconess Hospital has steadily declined as the total number of diabetic admissions has increased. Incidence of diabetic coma is summarized in table 2.

From this table, our conclusions have been that the decline in death rate has resulted chiefly from: (1) the earlier use of larger amounts of insulin, which is based on the belief that the first objective in the treatment of diabetic ketosis is to give insulin sufficient to obtain the optimal pharmacologic effect as rapidly as possible; (2) a better understanding and treatment of the electrolyte imbalance; and (3) better treatment of the truly emergency complications of diabetic ketosis which consist, first, of failing renal function with anuria and, second, of infectious complications.

Symptoms. No pathognomonic symptoms occur in early diabetic coma, but difficult respiration with the characteristic deep air hunger and drowsiness are outstanding evidences of an advanced stage. Actually, the symptoms of developing diabetic coma may be identified as follows: (1) urinary tract—with polyuria, increased glycosuria, and ketonuria; (2) intestinal tract—with loss of appetite, nausea, vomiting, and abdominal pain accompanied by constipation; (3) respiratory tract—with air hunger; and (4) central nervous system—with drowsiness and final coma.

Progression from early to later stages may advance slowly over a period of days or rapidly over a period of a few hours. In a fully developed diabetic coma, the patient is usually semiconscious or unconscious and completely relaxed, although, in some cases, there may be much activity. The skin—notably of the axillae and the tongue—is dry. The face is flushed and drawn; hands and feet are cold. Respiration may be deep. However, it must not be forgotten that the patient may have passed through this early stage of Kussmaul's respiration, and, during the state of exhaustion, the respiration may be extremely shallow. Eyeballs are soft to the touch. Care must be taken not to press quickly upon such eyeballs, for fear of separation of the retina. Despite the frequent vomiting, abdominal findings may include evidence of a distended, dilated stomach. Muscle spasm and abdominal pain may suggest acute appendicitis. The pulse is rapid and weak, and the blood pressure is low or even unobtainable. Rectal temperature is usually subnormal. If active infection is present, the fever of the hidden pneumonia, pyelonephritis, or phlebitis may return as the treatment proceeds.

TABLE 2
DIABETIC ACIDOSIS AND COMA
1,053 Cases Treated at the Joslin Clinic, Boston

Number of Cases	Average age by years	Duration of diabetes mellitus by years	Blood sugar CO ₂ mg. mm.	Insulin units	Per cent fatal
52 (1923-26)	31	3	480 6	161	18
411 (1927-39)	30	4	501 5	208	11
341 (1940-50)	29	7	536 6	302°	3.2
216 (1951-59)	30	9	559 6	328°	5.5
33 (1959-60)	30	8.3	633 7.5	333°	3.3

°268 cases received from 20 to 420 extra units (an average of 70 units) en route to the hospital.

DIFFERENTIAL DIAGNOSIS IN COMA

Drowsiness or unconsciousness constitute a serious emergency whether the patient is diabetic or nondiabetic. In a known diabetic, the possibility of acidosis and coma must be the first thought; the second must be possible hypoglycemia. One of the most fatal errors in all internal medicine is that of giving insulin to a patient who already has hypoglycemia.

1. *Hypoglycemia*. Patients in hypoglycemia are usually moist in contrast to patients in acidosis, but there are exceptions. Diabetic patients who have nephropathy with nitrogen retention may have most serious and even fatal hypoglycemia with dry skin and none of the usual symptoms. Mental symptoms in hypoglycemia may be profound, including confusion, delirium, and resistance. The symptoms from protamine zinc insulin in hypoglycemia are more gradual than with regular insulin and may require more prolonged treatment. When a patient is found unconscious a long distance from a laboratory, so that blood sugar tests are not available, a small amount of glucose in sterile solution may be given intravenously. If complete recovery is prompt, then an insulin reaction is probable. In most instances, however, transferring the patient to a hospital as an emergency case is a proper procedure.

2. *Uremia*. The problem of uremia is increasingly frequent, since more diabetic patients are living long. These patients are reaching the stage where, because of uncontrolled diabetes over a period of years, diabetic nephropathy is present. If albuminuria is marked, nitrogen retention and uremic symptoms may occur. The blood sugar level may be extremely high and the carbon dioxide (CO₂) content of the blood depressed as in diabetic ketosis, but the blood level for acetone will be normal. However, it is the pa-

tient with both diabetic ketosis, as shown by these findings, and renal failure, who will require careful laboratory study and great care in treatment.

3. *Cerebrovascular accidents*. These attacks may have a relatively sudden onset or may have been preceded by severe headache or some hours of symptoms. The spinal fluid may contain gross blood.

4. *Toxicity*. Acute infection, drug poisoning, brain tumors, or meningitis must be considered as possible causes of toxicity.

TREATMENT

1. *Hospitalization*. Diabetic ketosis should require hospital treatment in order to secure the benefit of laboratory and diagnostic procedures. It is customary to give insulin immediately at home provided the diagnosis seems certain. Actually, in our patients (summarized in table 2), doses ranging from 50 to 200 units have been given before admission to the hospital and as yet no serious errors, such as the administration of insulin to a patient in hypoglycemia, have been made. The decision is an important one and the size of the dose may be a matter of some question. The following outline lays emphasis upon treatment during the first and second hours.

OUTLINE OF TREATMENT OF DIABETIC ACIDOSIS AND COMA AT THE JOSLIN CLINIC

Diabetic acidosis is the result of insulin deficiency. Nausea, vomiting, abdominal pain, dehydration, shock, air hunger, and drowsiness lead to coma and, if insufficiently treated, death. Important chemical features are hyperglycemia, glycosuria, ketonemia, ketonuria, reduction of plasma CO₂, and depletion of electrolytes, especially potassium.

First hour after admission

Special nurse, preferably experienced in coma treatment, is needed for the first few hours.

LABORATORY

1. *Urine.* Examine for sugar, acetone, diacetic acid, albumin, coma casts, and pyuria. Catheterize if necessary.

2. *Blood.* Test for sugar, CO₂ content, and nonprotein nitrogen, with emergency report within an hour. Take white blood count. Determine serum potassium and amylase levels. Hematocrit reading aids in assessing dehydration.

CLINICAL

3. Search for complications and establish diagnosis.

A. Obtain history to explain cause of coma.

B. Give physical examination, noting particularly (a) state of consciousness, type of respiration, pulse rate, blood pressure, and rectal temperature; (b) soft eyeballs, dry tongue, dilated stomach, cold and mottled skin, impacted rectum, and tendon reflexes.

C. X-ray chest and abdomen when possible.

D. Make electrocardiogram to detect coronary and potassium changes.

4. *Insulin.* Give 50 to 100 mits of regular insulin (one-half dose intravenously) at once for adults. In severe cases, especially with circulatory collapse, give all insulin intravenously. If blood sugar exceeds 300 mg. per 100 cc. and if the blood CO₂ content is 9 mM. per liter (20 vol. per cent) or less, the dose will need to be repeated. The insulin dose would be proportionately less (20 to 40 mits) in young children, especially if diabetes is of recent onset. In cases with blood sugar between 600 and 1,000 mg., give 200 mits additional, and with blood sugar over 1,000 mg., give 300 mits additional.

5. *Gastric lavage.* Aspirate completely and wash stomach with warm water with greatest care.

6. *Normal saline.* Give 2,000 cc. intravenously. It is desirable to change to a solution of saline lactate after the first liter of saline solution is given. To 700 cc. of saline, add 1 ampule (40 cc.) 1 molar lactate and make up to 1,000 cc. with sterile distilled water. If lactate is unavailable, normal salt solution may be continued. Avoid too rapid administration, especially in older patients.

7. Keep patient warm yet avoid burns, as from a hot water bottle.

Second to sixth hour

The gravity of the case may require repetition of first hour's total insulin in the second hour.

8. Give potassium solutions by vein for definite indications: (a) when blood analysis or electrocardiogram clearly indicates hypokalemia; (b) when potassium depletion is probably present as a result of prolonged serious ketosis and/or deficient potassium intake; and (c) only in the presence of adequate urinary output, 25 mEq. of potassium per hour up to 100 mEq. may be given.

9. Repeat blood sugar and CO₂ determinations after two or three hours. For rising blood sugar, give insulin hourly 50 to 200 mits or more of insulin hourly, according to estimate of prognosis and hourly blood sugar tests.

10. Fluids are given by mouth as soon as tolerated and are limited to 100 to 120 cc. per hour of broth, ginger ale, orange juice, tea, or coffee to be sipped by patient or spooned by nurse. For children, limit fluids to 50 cc. per hour at first. Then, if nausea and vomiting recur,

withhold fluids orally for two to six hours. Lavage stomach again, if indicated, and then resume.

11. Give an enema to cleanse and to relieve abdominal distention.

12. Record and note changes in blood pressure, pulse, and temperature hourly. Consider vasopressor drugs or transfusion if patient is in deep shock.

13. Urinalysis for sugar and diacetic acid is performed every hour. Record hourly output as index of dehydration and renal function.

14. Parenteral antibiotics, such as penicillin, streptomycin, or tetracyclines, are frequently needed when blood pressure is normal.

15. Record urinary output hourly, and note with alarm any sign of oliguria. Give 1,500 cc. of intravenous saline lactate for persisting shock. Repeat as necessary. For anuria, associated with hypochloremia, give 50 cc. of 10 per cent salt solution intravenously. Beware of producing excessive diuresis with consequent loss of base, especially of potassium, by too rapid administration of intravenous fluids. For anuria, associated with hyperchloremia, omit all saline and use only glucose in water intravenously. Volume of such fluids may need to be limited to 1,000 cc. in twenty-four hours.

Sixth to twenty-fourth hour

16. Repeat blood sugar and CO₂ determinations. Give 50 to 200 mits of insulin if blood sugar and CO₂ levels are not improving. Regular insulin may be given according to urine tests every one to four hours if fall in blood sugar has been satisfactory.

If test is— Red Orange Yellow Green Blue

Give— 20 16 12 0 0 Units

For young children give half a dose.

17. Give soft or liquid food, such as oatmeal gruel, orange juice, or milk diluted half and half with lime water, not in excess of 10 gm. of carbohydrate per hour. Glucose, 5 per cent in saline, is given intravenously at the rate of 200 cc. per hour only when blood sugar approaches normal.

18. Sudden onset of muscular weakness, or loss of tendon reflexes, and shallow respiration suggest hypokalemia. Potassium may be given by mouth or intravenously if changes in the electrocardiogram or in serum potassium are present. See below, "Additional Notes."

Second day and succeeding days

19. Soft food diet is begun and consists of carbohydrate, 100 to 150 gm.; protein, 50 gm.; and fat, 50 gm. Gradually return to standard diabetic diet for age and weight of patient with carbohydrate, 150 to 200 gm.; protein, 60 to 100 gm.; and fat, 60 to 120 gm. daily.

ADDITIONAL NOTES

1. Differential diagnosis should include the acidosis of diabetic nephropathy occurring in patients with diabetes of long duration. Uremia may result in retention of ketone bodies in the blood plasma, although they may be absent or reduced in concentration in the urine. Examine plasma for acetone by nitroprusside test (4 cc. of blood in an oxalate tube is centrifuged until clear plasma is obtained. Make solutions of 1 in 2, 1 in 4, and 1 in 8 with normal saline or tap water. Place 3 drops of undiluted plasma and the 3 dilutions on separate small mounds of acetone test powder. At the end of sixty

seconds, read the color. Do not allow to stand longer. Depth of purple color indicates concentration of acetone and, in some cases, may be used as a clue to insulin resistance) or quantitate ketone bodies in blood.

Total Ketones in Blood

	Mg. per 100 cc.
Normal	0 to 5
Nondiabetic uremia	5 to 60
Diabetic coma	50 to 200+

2. To avoid pulmonary edema, rarely exceed 5,000 cc. of parenteral fluid in twenty-four hours and check frequently for signs of edema. If urinary output exceeds 40 cc. per hour after parenteral fluid has been given up to 3,000 cc., grave dehydration no longer exists.

3. Electrolyte-containing solutions: Potassium should not be given intravenously in excess of 25 mEq. per hour! It is rarely wise to exceed 100 mEq. in twelve hours unless definite hypokalemia is present and urine excretion is ample. After twelve to twenty-four hours, if 3 to 4 gm. of potassium cannot be taken by the patient on a diabetic diet, a simple solution may be taken in divided amounts. Thus, 200 cc. of orange juice plus 2 gm. of potassium phosphate may be diluted with water to 500 cc. Of this, give 100 cc. per hour. With fall in blood sugar and need for potassium, a 5-cc. ampule (2 gm. dibasic potassium phosphate and 0.4 gm. monobasic potassium phosphate) may be added to 1,000 cc. of 5 per cent glucose for intravenous administration if indicated.

4. Electrocardiographic signs of:

A. Low serum potassium (below 3 mEq.) consists of (1) lowered or inverted T waves, (2) depressed ST segments, (3) lengthened QT or appearance of U wave, and (4) prolonged P-R interval.

B. High serum potassium (above 6 mEq.) consists of (1) high, peaked T waves, (2) wide QRS, (3) disappearance of P waves, (4) atrioventricular dissociation, and (5) final disorganization of electrocardiogram.

Note. A normal electrocardiogram does not exclude K deficiency. The foregoing changes may not always be due to hypokalemia.

As soon as the diagnosis has been made and even before physical examination has been completed, a preliminary dose of insulin is given. In severe cases, we give at least 6 per cent of the initial dose intravenously. In the more severe types, we give all insulin intravenously during the first few hours. This insulin should be of the unmodified or regular type. Protamine zinc insulin acts so slowly that it should not be used except as an adjunct to treatment with rapid-acting insulin.

2. *Fluids.* Dehydration and loss of electrolytes are two of the most important clinical features of diabetic coma and, as indicated in the outline, the restoration of fluid electrolytes is begun immediately by intravenous administration of physiologic salt solution. Actually, the use of sodium lactate solution is almost as prompt as is indicated in the outline. In the average patient, from 3,000

to 6,000 cc. of fluid is necessary in the first six to twenty-four hours. During the first few hours of treatment, in the stage of hyperglycemia, no glucose solution is indicated.

In these first hours, the prime objective is to remember the presence or absence of insulin resistance, thereby securing the administration of a proper amount of insulin as quickly as possible. If the blood sugar level is maintained by glucose administration, then the decline in blood sugar may not be used as an index of the insulin requirement. If levulose is used, it is possible for serious hypoglycemia to be produced by giving too much insulin. This can happen because the presence of levulose in the blood will give a high blood sugar value, although the "true" glucose value of the blood may be at a seriously low level. When glucose is being used in conjunction with large amounts of insulin, it is important that the quantity of glucose administered will directly affect potassium — the more glucose that is given the more potassium will be withdrawn from the blood and deposited with the glycogen. Potassium is administered in comatose patients when the history indicates with certainty that depletion of potassium is present, whether because of long-continued lack of diabetic control, or because of acidosis itself. It is also administered when serum potassium values or the electrocardiogram indicates the need.

3. *Gastric lavage.* An important measure in preparing the gastrointestinal tract to receive food, which is an objective in the treatment of coma, is the use of gastric lavage. It should be carried out routinely unless the patient is in such grave condition that the procedure involves danger. Usually one is surprised by the finding of considerable quantities of fluid, food remains, and old blood in the gastric content.

4. *Glucose and food.* The use of glucose depends upon which period in the treatment of diabetic coma is being considered. The three- to ten-hour period after admission is characterized by hyperglycemia and dehydration. Administration of glucose tends still further to dehydrate the intracellular space. However, when the blood sugar has begun to decline and has reached roughly one-third to one-half the original level, the administration of nutrient is important. Then glucose solution may well be given. At the Deaconess Hospital, we have at times used glucose for twenty-four to forty-eight hours or more in seriously comatose patients. It is used after the hyperglycemia has been controlled and when the persisting brain damage makes it impossible to feed the patient by mouth.

5. *Other remedies.* Transfusion has been occasionally useful in a patient with severe shock and persisting low blood pressure. The use of antibiotics and vitamins is common for unconscious patients who may be suffering from hidden infection and who may, because of severe metabolic disturbances, be in danger of developing diabetic neuropathy.

6. *Convalescence.* Recovery from acute acidosis may be followed by a return to normal which is steady but prolonged. It is important to remember that patients who have been unconscious for long periods or who have been in severe shock may not be able to take much food or return to a full diet for a week or two.

COMPLICATIONS

Circulatory collapse with low blood pressure, rapid pulse, and oliguria may occur as a late event. Anuria or oliguria is an ominous complication in ketosis. It is essential to chart the urine output of the patient in diabetic coma hour by hour and to be alarmed by any decline in the rate of urine secretion. Then determinations of serum electrolytes, with reference to potassium and sodium, may give the clue for therapy. Anuria enduring for days may still terminate in recovery.

CASE REPORT

Martha N., aged 12, with newly discovered diabetic coma, was referred to the Deaconess Hospital in anuria. Her original blood sugar was 435 mg. per cent with a CO_2 of 11 mEq./l. When anuria persisted, she was finally transferred to the Peter Bent Brigham Hospital, Boston, for dialysis by the artificial kidney. This was successful in restoring urinary secretion. Then a renal biopsy disclosed the findings characteristic of healing acute tubular necrosis. A tragic effect of her diabetic ketosis was that within a few weeks her eyegrounds showed the typical dilated veins of early diabetic retinopathy. Within a few months, generalized edema of

the fundi with striate hemorrhages and two new capillaries was noted, indicating retinitis proliferans and moderate sclerosis of the arterioles.

Other causes of anuria seen in our cases of diabetic coma were: (a) dehydration and circulatory collapse—the most frequent cause; (b) acute or chronic pyelonephritis; (c) acute renal injury, as in necrotizing papillitis or the hepatorenal syndrome; (d) acute glomerulonephritis, often indicated by history of facial edema and smoky urine; (e) shock from burns or trauma; (f) congenital absence of kidney; (g) obstruction from stones, sulfonamide crystals or tumor; and (h) acute renal shutdown, a rare event occurring even in early ketosis.

The treatment for acute renal shutdown involves the avoidance of excessive fluid administration by carefully limited intravenous feedings and by careful administration of electrolytes, sometimes for prolonged periods.

APPENDIX

Diabetic ketosis and coma

Definition. An intoxication caused by the accumulation of excess products of intermediate protein and fat metabolism, resulting primarily from insulin deficiency. Drowsiness and coma represent reaction of the brain to ketosis, dehydration, acidosis, and mineral depletion.

Etiologic factors: (1) insufficient or omitted insulin, (2) infections, (3) resistance to insulin, (4) anesthesia and shock, (5) thyrotoxicosis, and (6) pregnancy and toxemias.

Dietary errors are the most frequent etiologic factors.

Pathophysiology. With insufficient insulin, a rise in blood sugar level and glycosuria occur. Loss of calories stimulates mobilization of protein and fat. Fatty acids are converted in the

TABLE 3
WATER AND ELECTROLYTE DISTRIBUTION IN A 70 KG. (154-LB.) MAN*

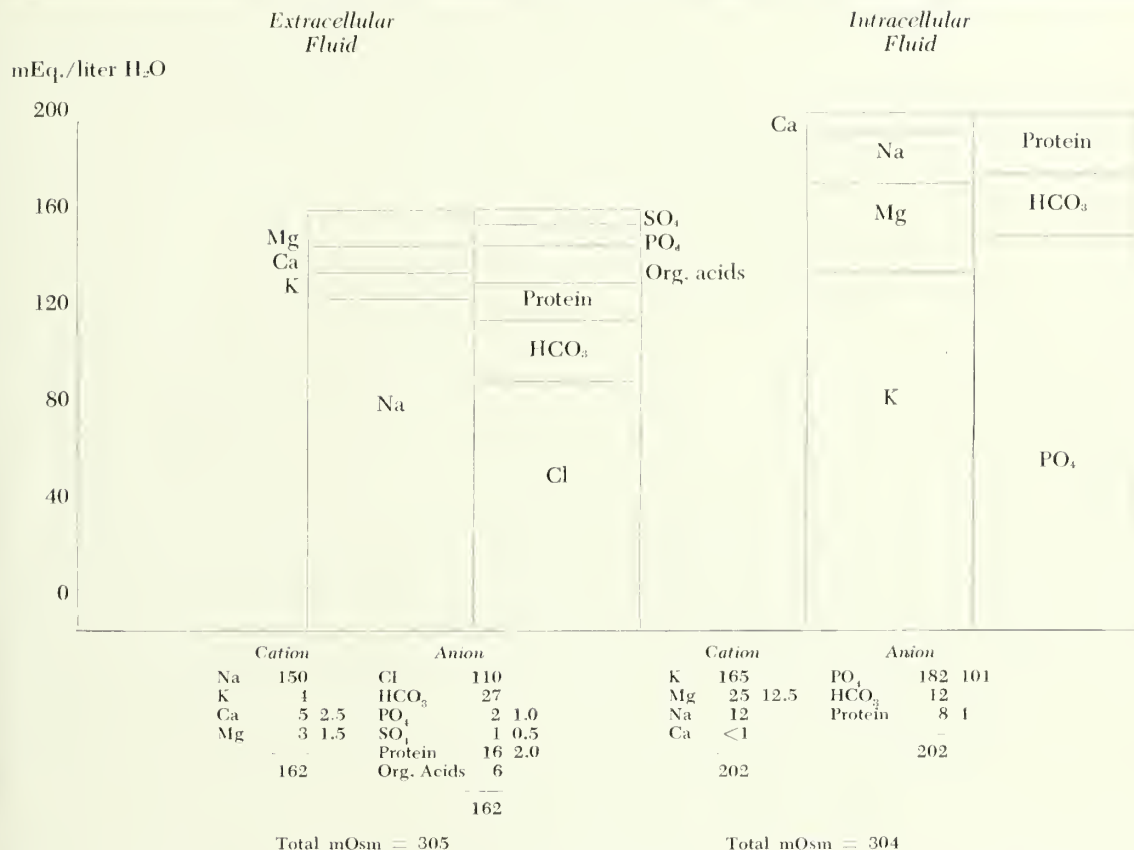
	H ₂ O		Na		K		Ca		Mg		Cl		PO ₄	
	Kg.	Per cent Total	mEq.	Per cent Total	mEq.	Per cent Total	mEq.	Per cent Total	mEq.	Per cent Total	mEq.	Per cent Total	mEq.	Per cent Total
Interstitial														
ECW ¹	10.5	21	2,100	60	56	1	70	1	42	2	1,680	100	28	<1
Plasma														
	3.5	7												
ICW ²	35.0	72	385	11	5,600	99	702	1	944	39			3,540	6
Bone			1,000	29			124,500	99	1,409	59			52,100	94
Total	49.0	100	3,485	100	5,656	100	124,640	100	2,395	100	1,680	100	55,668	100

¹ Extracellular water

² Intracellular water

*NICHOLS, N., in JOSLIN, ROOT, WHITE, and MARBLE. *Treatment of Diabetes*, ed. 10 Philadelphia: Lea & Febiger, 1959, p. 378.

TABLE 4
ELECTROLYTES OF EXTRA CELLULAR AND INTRACELLULAR FLUID*



*NICHOLS, N., in JOSLIN, ROOT, WHITE, and MARBLE: Treatment of Diabetes, ed. 10. Philadelphia: Lea & Febiger, 1959, p. 378.

liver to ketone bodies. As metabolites are formed in excess, blood levels rise and exceed the renal threshold. The serum carbonate falls as base is excreted. Ketone bodies accumulate as renal function fails.

WATER AND ELECTROLYTES

1. Normal maintenance of body fluids and electrolyte balance is accomplished by the constant interplay of a number of mechanisms. In table 3, the distribution of water and electrolytes is shown in a normal man. In table 4, the relative composition of extracellular and intracellular fluids is indicated. The following are effective mechanisms in maintaining normal distribution:

A. Nutritive—normal diet provides excessive water and electrolytes which are selectedly retained or eliminated.

B. Hormonal—ions are retained for intracellular needs.

C. Physiochemical relations occur at cell membranes.

D. Renal variations exist in secretions and absorption of electrolytes and in the manufacture and substitution of ammonia and hydrogen ions.

2. Initial metabolic defects in acidosis are as follows:

A. There is a cessation of normal intracellular carbohydrate metabolism.

B. Acetate fragments from fat metabolism are condensed to form diacetic acid which finally appears as the 3 acetone bodies in the plasma. Fat changes to CO_2 and water in tricarboxylic acid cycle. For this entry, two-carbon acetyl CoA or "active acetate" from fat must combine with oxalacetate derived from carbohydrate or protein. Lack of carbohydrate oxidation and failure of protein to supply oxalacetate explain the accumulation of acetate fragments.

TABLE 5
LOSSES OF WATER AND ELECTROLYTE DURING DIABETIC COMA IN A 70+ Kg. MAN^o

	ECW ¹		ICW ²		Total†	
	Gm.	Per cent Total	Gm.	Per cent Total	Gm.	Per cent Total
H ₂ O	3810	27	3830	11	6866	14
	mEq.		mEq.		mEq.	
Na	591	28	26	7	531	15
K	7.8	14	490	9	493	9
Ca	—	—	—	—	252	.002
Mg	—	—	58	6	56	2
Cl	420	25	—	—	430	26
PO ₄	—	—	—	—	344	.006

¹Extracellular water

²Intracellular water

^oNICHOLS, N., in JOSLIN, ROOT, WHITE, and MARBLE: *Treatment of Diabetes*, ed. 10, Philadelphia: Lea & Febiger, 1959, p. 378.

†The values are averages compiled by the author, N. Nichols.

C. With glycogen and protein breakdown, glucose, nitrogen, water, and electrolytes are released into the extracellular space. These with ketones increase the osmolarity of plasma; extracellular body increases. The increase in plasma volume leads to increased glomerular filtration, less reabsorption by proximal renal tubules, and polyuria. There is excretion of acids as well as electrolytes.

3. Homeostatic defense mechanisms (compensated acidosis):

- Buffers, bicarbonate. Extracellular bicarbonate is chief.
- Hormonal responses.
- Renal defense.
- Body base stores.

Ratio of H₂CO₂ to BHCO₃ (1:20) is changed by the loss of base (B), used to neutralize ketones. The ratio of acid to base may increase. Hydrogen ion concentration (pH) (7.38–7.42) may decline.

The stress response affects the hypothalamic-pituitary-adrenal-axis. This response is unfavorable so far as carbohydrate is concerned, since the blood sugar is raised and carbohydrate metabolism is inhibited. But the effects of aldosterone-like substances conserve sodium, although potassium is excreted through their effect upon renal tubules. The physiologic action of the antidiuretic hormone in coma is not well understood. Conservation of water by the excretion of a concentrated urine does not occur in ketosis.

Kidney. There is increased secretion of ketones, organic acids, and so on in order to pre-

serve normal body concentrations. Hydrogen is elaborated to excrete organic acids without base. An increased ammonia production conserves base as well as induces a change from dibasic to monobasic phosphate with consequent retention of sodium. Ammonia replaces base. Nevertheless, sodium (Na) is lost even from bone. This loss is compensated so long as pH does not fall.

4. Uncompensated severe ketoacidosis is indicated by CO₂ of less than 9 mEq. Lower values mean an exhaustion of buffering systems. Changes in pH follow. The intracellular pH is lower than the extracellular pH. A pH below 6.8 is frequently fatal, since below this level intracellular metabolism ceases. But recovery with a pH of 6.8 is recorded.

Losses of water and electrolytes. The extreme catabolism of body tissues results in accumulation in the extracellular space of nitrogen, ketones, and electrolytes, especially potassium. Excretion results in losses of water and electrolytes during severe coma as H₂O intake ceases, and contraction of extracellular space, hemoconcentration, decline in plasma volume, and fall in blood pressure occur (table 5). Then renal ischemia, granular casts, and tubular necrosis follow.

5. Replacement therapy consists of three phases:

- Maintaining water and the electrolyte balance.
- Sustaining fluid volume and maintaining normal urine output. Here potassium (K) is important.

C. Convalescing—a phase for correction of intracellular needs. The only contraindication to K therapy is oliguria.

UNITS OF MEASUREMENT OF BLOOD ELECTROLYTES

The figure for milliequivalents per liter was obtained by use of the following formula:

$$\frac{\text{No. of mg. per 100 cc.} \times \text{valency}}{\text{Atomic weight}} \times 10 = \text{milliequivalent per liter}$$

Example. Conversion of plasma chloride value of 355 mg. per 100 cc. to milliequivalents as follows:

$$\frac{355 \times 1}{35.5} \times 10 = 100 \text{ mEq. per liter}$$

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CHANGES IN THE exocrine glands of the gastrointestinal mucosa due to mucoviscidosis predispose to peptic ulcer. Mucosal cysts erode under the influence of external irritants and acid gastric juice.

Functional disturbance of exocrine glands with pathologically viscid mucus occurs not only in infants and children but also in adults. Important manifestations are excretory pancreatic insufficiency, bronchiectasis, pulmonary emphysema, high sodium and chloride content of sweat, electrolyte depletion, and low blood pressure. The disease follows an autosomal dominant mode of inheritance.

Adult mucoviscidosis is particularly likely in middle-aged men with duodenal ulcer having (1) unexplained cachexia, dehydration, low blood pressure, and a tendency to collapse; (2) several ulcers; or (3) pulmonary emphysema since childhood, with cyanosis of the lips and clubbing of the fingers. Discovery of high sodium and chloride content of sweat or relatives with mucoviscidosis confirms the diagnosis.

Of 41 adults with mucoviscidosis, 17 had active or healed gastric or duodenal ulcers. Incidence of mucoviscidosis with peptic ulcer was high among relatives of the patients having mucoviscidosis with or without ulceration. Of 43 unselected ulcer patients, 4 had all the cardinal manifestations of mucoviscidosis and approximately half had some features of the disease. Of 32 healthy persons and 88 hospital patients with various diseases, all had normal sweat levels of sodium and chloride and normal pancreatic enzyme activity.

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Diabetic Pregnancies in a Community Hospital

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PHYSICIANS ARE APPALLED and challenged by the problem of coexisting pregnancy and diabetes. The problems of fetal hazards and secondarily of maternal hazards, although not thoroughly understood, are widely recognized. Our purposes in this study were to appraise our results, to find what changes should be made in our treatment, and to see whether results have been achieved comparable to those reported by larger institutions.

METHODS

This series is a retrospective study of 45 pregnancies in 31 patients, which were completed in St. Luke's Hospital, Fargo, between July 1, 1953, and August 30, 1959. Patient ages ranged from 20 to 45 years. Some of these patients reported to their obstetricians for the first time late in pregnancy and had little or no medical care prior to delivery. In the majority, however, a complete history and physical examination were carried out at the first visit, usually early in the pregnancy.

Sufficient diagnostic studies were completed early in the pregnancy to classify the pregnancy risk according to the method of White¹ (table 1). Such a classification is essential to a report on this subject because of the clinical differences, both apparent and subtle, between patients and the differences in outcome particularly to the fetus.

Visits were arranged to coincide with visits to the obstetrician—monthly early in the pregnancy, later biweekly and weekly during the last month. After the first visit, laboratory work was kept at a minimum, consisting of a urine

test for albumin and sugar, a blood test for hemoglobin at each visit, and nonfasting blood sugar tests—usually 3 or 4 during the course of the pregnancy.

Quantitative diabetic diets were prescribed using the exchange system. Some patients weighed their food; however, the majority used household measurements. Caloric allowances varied between 1,500 and 2,000. Protein allowance was approximately 1 gm. per kilogram of body weight. Carbohydrate-fat ratios varied from 1.5 to 2 gm. of carbohydrate per gram of fat. Usually, sodium was not restricted in the case of class A patients. Those in classes B through F were usually allowed 800 to 1,200 mg. of sodium. A few were restricted to as little as 400 mg.

All patients took a single morning insulin injection and were told in detail how to adjust the insulin dose to maintain good urine tests. The largest single group of patients used only NPH insulin. A few used lente insulin. A few used a mixture of NPH and crystalline insulin or lente and crystalline insulin, with the insulins mixed in the syringe, the ratio being varied by the patient according to previous instructions. One patient used globin insulin. All patients received a prenatal capsule containing vitamins iron, and calcium three times daily. Diuretics were begun only when evidence of edema or hydramnios was found, and then they were continued through the rest of the pregnancy. No hormone treatment was used in any of these patients. Each patient was required to test her urine for sugar before meals and at bedtime and to bring a record of these tests, her daily insulin requirement, and her insulin reactions to the physician at each visit. Emphasis was placed on strict chemical control of the diabetes; how-

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ever, this was not always achieved to the extent desired.

Most class A patients delivered at or near term by the vaginal route. The remainder of the patients were delivered at thirty-two to forty weeks, the majority of these between the thirty-sixth and thirty-eighth week. Eight cesarean sections were done, half of these for purely obstetric reasons. The delivered infants were under the care of qualified pediatricians. Routine treatment consisted of immediate aspiration of the respiratory tract and stomach, close observation for respiratory distress, and initiation of oral feedings after forty-eight hours. All infants were kept in the incubator for the first few hours and longer if necessary. For the most part, oxygen was used only as indicated by respiratory distress.

RESULTS

Table 2 tabulates our experience with 45 diabetic pregnancies over a six-year period. The diabetic condition in all of the 9 patients in class A was discovered during pregnancy or at the time of delivery. At least 3 of these are pre-diabetics, since they had normal glucose tolerance tests following delivery. Presumably most or all of the remainder are also. These 9 patients underwent a total of 13 pregnancies, resulting in 13 living, healthy infants.

In class B, 7 patients had 12 pregnancies. Of these, 9 survived to viability, for which we used gestation of twenty-eight weeks as our definition. Of these, 8 infants were delivered alive and 7 of them survived, resulting in 58 per cent successful pregnancies and a fetal survival rate of 78 per cent.

In class C, 9 patients underwent 12 pregnancies, resulting in 10 live births, 8 of which survived, giving 67 per cent successful pregnancies and a fetal survival rate of 80 per cent.

TABLE 1
DIABETIC PREGNANCY RISK CLASSIFICATION
(WHITE¹)

Class
A. Borderline diabetics requiring no insulin
B. Duration under 10 years and onset over age 20; no vascular abnormalities
C. Duration 10 to 19 years or onset age 10 to 19; no vascular abnormalities
D. Duration over 20 years, onset under age 10, calcified leg arteries, or retinitis
E. Calcified pelvic arteries
F. Diabetic nephropathy

TABLE 2
45 DIABETIC PREGNANCIES
JULY 1, 1953, TO AUGUST 30, 1959

Class	A	B	C	D	F
Patients	9	7	9	5	1
Pregnancies	13	12	12	7	1
Viable	13	9	10	6	0
Live births	13	8	10	5	0
Surviving infants	13	7	8	3	0
Successful pregnancies	100%	58%	67%	43%	0%
Fetal survival	100%	78%	80%	50%	0%

In class D, 5 patients underwent 7 pregnancies, 6 of which reached viability. Of these, 5 progressed to live births and 3 of the infants survived, giving 43 per cent successful pregnancies and a 50 per cent fetal survival rate.

No class E patient was seen in this series.

One class F pregnancy was seen which did not progress to viability.

DISCUSSION OF LOSSES

There was no maternal mortality in this series. Table 3 tabulates our fetal losses by stage in each class. Preivable losses numbered 7. One class B patient accounted for 2 of these. The first of her pregnancies was complicated by severe Graves' disease and bilateral subtotal thyroidectomy at thirteen weeks. In the twenty-seventh week, she was admitted in ketosis with a CO₂ of 29 volumes per cent and, on the following day, spontaneously delivered a dead fetus with multiple congenital anomalies. One year later in the presence of recurrent hyperthyroidism, she became pregnant and a therapeutic abortion was done. There were 4 spontaneous abortions at eleven to thirteen weeks' gestation, 1 in class B, 2 in class C, and 1 in class D. A single class F patient had a therapeutic abortion at twelve weeks because of malignant hypertension and uremia.

Stillbirths occurred in 2 pregnancies. One such

TABLE 3
LOSSES

Class	B	C	D	F
Pregnancies	12	12	7	1
Preivable loss	3	2	1	1
Stillbirths	1	0	1	0
Neonatal deaths	1	2	2	0

birth was delivered by a class B patient whose diabetes was badly controlled throughout the pregnancy and who had ketosis with a CO_2 of 22 volumes per cent in the third trimester. At thirty-five weeks, she spontaneously delivered a macerated fetus. One class D patient underwent a cesarean section at thirty-nine weeks because of a contracted pelvis and mild toxemia. Heart tones were poor before surgery, and the infant was dead on delivery. Pathologic examination of the placenta showed chronic vasculitis of the decidual portion. Possibly, delivery one to two weeks earlier would have been more successful.

There were 5 neonatal deaths. One in a class D patient was due to a tracheoesophageal fistula and intracranial hemorrhage. One class C patient had spontaneous premature labor at thirty weeks. Autopsy on the infant showed no specific findings, and presumably death was due to prematurity. One class C patient with severe sprue underwent a cesarean section at thirty-two weeks because of a previous stillbirth. At autopsy, the infant weighing $7\frac{1}{2}$ lb. showed posthemorrhagic necrosis of the adrenals and hyaline membrane disease. It is questionable whether the loss might have been avoided by later delivery. One class B patient underwent cesarean section at thirty-four weeks, and a class D patient had mechanical induction and vaginal delivery at thirty-five weeks. Autopsy on these 2 infants showed hyaline membrane disease. Possibly, these losses might have been avoided by later delivery.

COMMENTS

Reported results^{3,4} in diabetic pregnancies have varied greatly, especially when one considers the very good results reported by Stephens⁵ and by White.² In appraising our results, aside from the obvious factors of patient cooperation and professional competence, we developed the following impressions:

1. A pregnancy risk classification is essential, since it foretells the basic statistical risk upon which individual factors are superimposed.

2. Serious intercurrent disease of the mother, especially during the pregnancy, increases the risk of failure.

3. In the majority of cases, the advantage of

elective early delivery is doubtful and must be carefully weighed against the risk to the infant of prematurity. We propose the following broad principles for our own future guidance. We shall deliver at term all class A patients and the more favorable class B patients. All those remaining will be delivered at thirty-eight weeks or as soon thereafter as induction and vaginal delivery can be accomplished, with the exception of patients who have had previous fetal losses at thirty-seven weeks or later. These patients we propose to deliver at thirty-seven weeks, either vaginally or by cesarean section if necessary. Generally, cesarean section will be reserved for these latter patients and for obstetric indications, including toxemia and very large babies.

4. We believe in the best possible control of diabetes at all times. In this series, possibly 2 failures were due to grossly poor diabetes control. However, the best control does not remove the risk imposed by some of the factors previously mentioned.

CONCLUSIONS

The diabetic woman embarking on a pregnancy faces a serious likelihood of disappointment. To a considerable extent, her chances of success are determined prior to conception by (1) her physical condition and (2) the duration of her diabetes and the age at which it began. However, we have the means at hand to improve the possibilities of success by assiduous medical and obstetric care of the mother during the pregnancy and delivery and by pediatric care of the newborn infant.

Good results may be obtained in the community hospital by means of the closely integrated efforts of internists, obstetricians, and pediatricians.

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Primary Malignant Mesothelioma of the Pleura

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THE DIAGNOSIS OF primary malignancy of the pleura continues to be quite a difficult problem in spite of all modern clinical skills and advanced laboratory and x-ray facilities. Unfortunately, this disease is quite uncommon, and few investigators have been able to accumulate a broad knowledge of its characteristic features. Nevertheless, mesothelioma is perhaps not as rare as the literature reports and probably may be seen but not recognized properly.

This tumor is most frequently confused with benign lesions of the pleura or the mediastinum, with primary malignancy of the underlying lung tissue as well as of the enclosing rib cage, and, finally, with metastatic disease from a distant tumor. Many diagnostic difficulties are created by the great variety of clinical, roentgenologic and pathologic manifestations of this tumor that may puzzle clinicians, chest surgeons, roentgenologists, and pathologists. This peculiar behavior was explained at first by Maximow¹ and later by Stout and Murray² with the aid of cell culture methods.

The growth originates from mesothelial (celomic) cells that are multipotential and can form a great variety of mesothelial as well as mesenchymal tissues in manifold combinations.³ Thus, mesotheliomas not only differ from each other but also may show amazing differences in microscopic sections of the same growth.³

Clinically, 2 types can be distinguished: a localized and a diffuse mesothelioma. The localized form is usually benign, fibrous, and asymptomatic until late and is often discovered during routine chest x-ray examinations. It forms a globular density attached to the chest wall or the diaphragm. There are all kinds of transitions between this tumor and the malignant types which grow diffusely, invade rapidly, and show early symptomatology. The latter may be respon-

sible for chest pain and discomfort before being visible on roentgenograms. Later, they make themselves known by pleural effusions, idiopathic pneumothorax, or dense massive shadows covering a large portion of the hemithorax.

Microscopically, the benign mesotheliomas form fibrous masses, while those that are malignant consist either of solid conglomerations or glandular and follicular arrangements of epithelial cells or, more rarely, of fibrosarcomatous tissue. The localized tumors can be eradicated surgically; the diffuse types, however, have been considered inoperable until recently when Harris and associates⁴ reported a cure, or at least a long-term survival, after radical pleuropneumectomy during the early stage of the development. In addition, Richert and Sherman⁵ reported a long-term arrest after early administration of radioactive gold.

If the experience of these investigators can be confirmed by others, it seems mandatory for all physicians who may encounter mesotheliomas in their practice to acquaint themselves with the symptomatology and natural history of this growth. It is obvious that a tumor of such great variability will produce a different picture in each individual case. However, the experience gained from the observation of 2 patients with malignant mesothelioma showed a characteristic similarity that makes it worthwhile to review them. The first case was previously reported in detail,⁶ while the second is a new case.

CASE REPORTS

Case 1. A 57-year-old oil refinery foreman noticed a diffuse pain in his left upper chest and upper abdomen, which gradually increased in intensity. The onset was very insidious, and his initial discomfort was at first not clearly separated from a previously present angina pectoris, in spite of the fact that the chest pain had changed in character and persistence and no longer responded to vasodilating remedies.

Initially, an x-ray film of the chest was normal, but soon this patient experienced a "spontaneous" pneumothorax without a history of trauma or physical exertion. A roentgenogram taken at this time showed a partly collapsed lung without abnormal shadows in this organ, in

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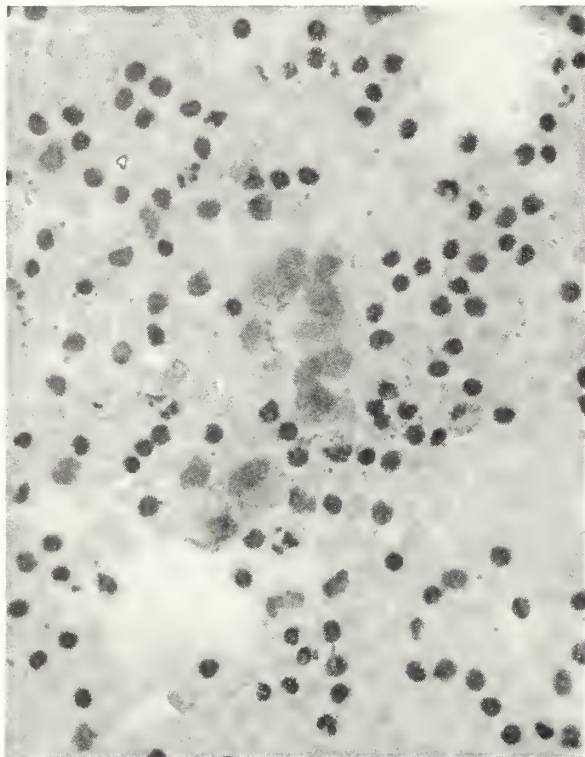


Fig. 1. Large mesothelial cells noted in the bloody pleural effusion.

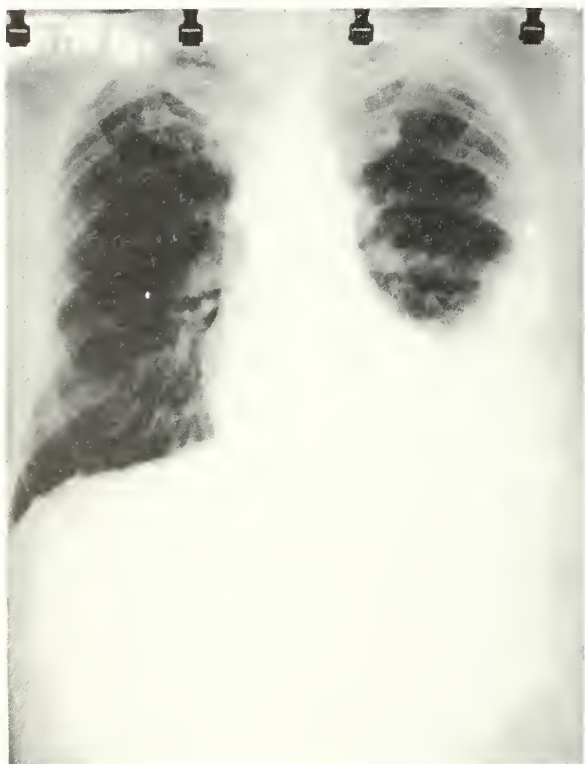


Fig. 2. Upper scalloped border of the pleural walls.

the pleural space, or in the bony rib cage. A small amount of serous fluid was aspirated but not examined for tumor cells. The chest pain persisted after the air had been absorbed, and a dense fibrothorax gradually developed with shrinking of the entire hemithorax and inward retraction of the lateral chest wall.

Thoracic exploration was performed because of unbearable pain, but this revealed only a dense fibrothorax with massive adhesions. Decortication gave no relief. Anorexia and weight loss became marked and led to severe cachexia. Neither large doses of narcotics nor neurosurgical procedures reduced the pain. An exploratory laparotomy followed, but only similar adhesions were encountered. No diagnosis was made until a routine stomach x-ray film unexpectedly revealed that several ribs were destroyed. Biopsy of this region revealed pleural mesothelioma.

Case 2. A 58-year-old oil refinery foreman who had worked with asbestos insulating material for quite a while noticed soreness and a rattling noise in the left hemithorax. Chest examination in January 1959 revealed a bloody pleural effusion. This fluid was bacteriologically negative but contained large mesothelial cells, which our pathologist suspected were malignant (figure 1). However, his suspicion was not shared by a number of other pathologists who were consulted. The patient's pain continued to increase and radiated over the whole left hemithorax. The pleural fluid reaccumulated in spite of repeated thorough paracenteses. Some dyspnea but little cough was noted.

Exploratory thoracotomy in a large medical center revealed multiple hard plaques covering the entire pleural space and extensive pleural adhesions. A portion of the pleura was removed for microscopic studies. The pathologist reported "granulomata of unknown origin." A lung biopsy performed at the same time revealed asbestos bodies in the bronchioles. In spite of negative skin and bacteriologic tests, the patient was placed on an anti-tuberculous regime after the operation. The fluid did not return, but a dense fibrothorax developed with shrinking of the entire hemithorax. The pleural density increased on successive x-ray films and finally revealed an upper scalloped border (figure 2). The pain was constant day and night and did not respond to large doses of narcotics.

In November 1959, intercostal blocks were performed followed by nerve sections. These procedures were without benefit, and, in December 1959, a chordotomy was also done. Shortly after this operation, x-ray examination revealed that several ribs were destroyed. Similar roentgenograms had been previously made almost at monthly intervals, but no bony defect had ever been noted. Excisional biopsy of these bones revealed a malignant growth interpreted as "fibrous sarcoma" by the pathologist (figures 3 and 4).

In the following weeks, various other parts of the bony thoracic cage were destroyed, particularly the lower dorsal vertebrae and the upper sternum. This led to cord compression and transection as well as to obstruction of the trachea and esophagus. The patient suffered unbearable pain until his demise in May 1960.

The essential findings at autopsy were "Mesothelioma of the left pleura invading mediastinum, ribs, vertebrae, liver, spleen, and lungs. Hypostatic pneumonitis, asbestosis of the lungs. The primary tumor and the metastases consisted of irregular bundles of spindle cells of atypical character with hyperchromatic bizarre nuclei" (figures 3 and 4).

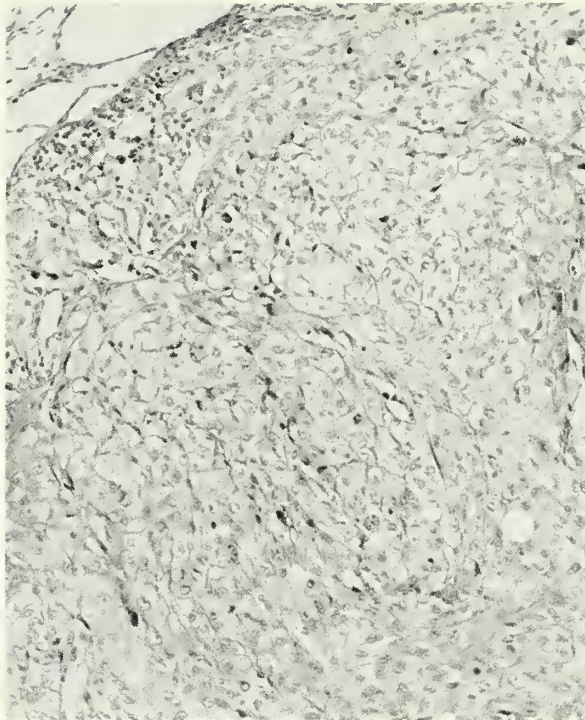
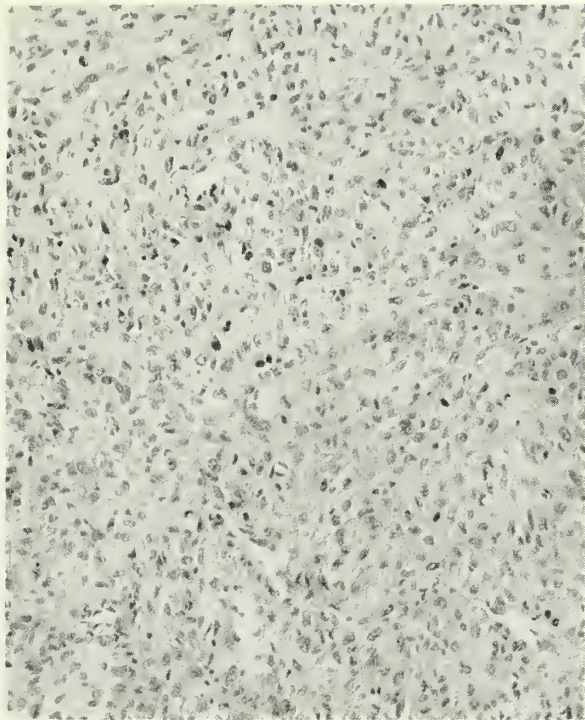


Fig. 3 (left) and Fig. 4 (right). Fibrosarcoma composed of irregular bundles of spindle cells with hyperchromatic bizarre nuclei.

DISCUSSION

The similarity of these 2 cases of diffuse malignant mesothelioma and of some of the others reported in the literature,⁷⁻¹⁰ is striking. Only the lack of familiarity with this entity can explain why both of the medical centers caring for these patients failed to make an early diagnosis, in spite of the fact that the referring physician alerted them to the possibility of such a growth. These patients were observed for months by various specialists who failed to recognize this entity until bone destruction, which was noted on roentgenograms, and excisional biopsy confirmed the correct diagnosis.

Any attempt to give these patients the benefit of radical surgery or successful radiation therapy would require a thorough knowledge of the early symptomatology of this tumor. The clinician must be alerted to a diffuse unilateral chest pain of recent origin gradually increasing in intensity in a middle-aged or elderly person. His physical examination may be negative, or there may be an unexplained pleural effusion and, occasionally, a spontaneous pneumothorax. The bacteriologic examination of the pleural fluid will be negative. It remains to be seen whether the cells present in this fluid can be recognized as malignant by the pathologists (figure 1).

The pain is not markedly relieved by para-

centesis or aspiration of the pneumothorax. Anorexia, weight loss, and cachexia gradually develop. Administration of narcotics and neurosurgical procedures are without benefit or only slightly useful to the patient. Percussion density and shrinking of the hemithorax, with or without scoliosis, may be observed. Clubbing of fingers, articular rheumatism, and osteoarthropathy have been reported in some cases but have not been observed in our patients. They are apparently more often seen with the localized benign mesotheliomas. Chills, fever, cough, dyspnea, and cyanosis are usually mild or absent.

The roentgenologist should be aware of the fact that the chest x-ray may be entirely negative for some time. Pleural effusion, fibrothorax or pneumothorax are nonspecific. However, the increasing density of the fibrothorax, particularly after surgical exploration and the shrinking and narrowing of the entire hemithorax may be significant. Either the mediastinum is pulled toward the lateral chest wall or vice versa. Scalloped margins of the pleural walls or of the fibrothorax (figure 2) are late manifestations. Bone destruction should be constantly looked for with repeated Bucky exposures. If present, this announces the final, probably incurable stage.

Thoracic exploration is always necessary to confirm the diagnosis. Therefore, the chest sur-



Fig. 5. Calcium deposit along the diaphragmatic surface of the pleural space.

geon must be familiar with the various macroscopic features of the growth which forms fine nodules, large plaques, massive adhesions, and a dense fibrothorax.

Of utmost importance is, of course, the correct pathologic interpretation of the pleural biopsy on which the decision for radical treatment depends. In the majority of cases, the specimens have been misinterpreted as fibrous pleural thickening, pleural adhesions, granulomatous tissues, or metastatic malignancy. The careful pathologist can only state that the lesions are compatible with malignant mesothelioma, because this diagnosis actually requires a complete autopsy excluding any other primary neoplasm. However, in practice, one should proceed with surgical therapy if a thorough clinical investigation has eliminated any distant malignancy.

Our second case is particularly interesting because of the history of long-time exposure to asbestos and the discovery of asbestos bodies in the lung biopsy specimen. The etiologic association of asbestos and malignant mesothelioma has been repeatedly discussed in the literature.¹¹⁻¹⁵ Not all investigators agree that exposure to asbestos predisposes to malignancy of the pleura.¹⁵ However, such history alerted the suspicion of the authors in the second case.

On the basis of his autopsy, our pathologist

considered it "very unlikely that the pathogenesis of the tumor could be related to the asbestos fibers since they were located in the bronchioles and not in the pleura." However, asbestos material could have reached the pleural tissues without being demonstrable microscopically.¹¹ In addition, along the diaphragmatic surface of the pleural space there was a "thick grey rind which was partially calcified" at autopsy. This calcium deposit could be demonstrated on the initial x-ray pictures (figure 5, double arrows), indicating some pleural scarring prior to the development of the malignant growth.

SUMMARY

The symptomatology and natural course of primary malignant mesothelioma has been illustrated by 2 case reports. In spite of early clinical suspicion, early thoracic exploration, and continuous care by various specialists, these patients progressed to an advanced hopeless stage with destruction of the bony thorax before the correct diagnosis was established. The early clinical, roentgenologic, and pathologic characteristics of this neoplasm must be kept in mind in order to bring these patients in time to radical surgical procedures or effective irradiation therapy that may be curative or at least prolong life.

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The Comatose Patient

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COMA REFERS to a state of complete or almost complete loss of consciousness from which the patient cannot be aroused by ordinary stimuli. In many cases, it comprises a definite medical emergency that must be evaluated rapidly and treated promptly to obtain the best results. In the absence of a good history, the formulation of the proper diagnosis may indeed tax the ability of the physician and will require a broad fund of medical knowledge and a considerable measure of intellectual resourcefulness. Often, even in the absence of an adequate history, careful attention to the significance of various clinical findings, along with well-chosen laboratory procedures, will be adequate to suggest the correct diagnosis.

Probably the best aid to the physician in evaluating a case of coma is an accurate knowledge of the most common etiologies of this condition. One of the most helpful methods of classifying coma is by the frequency of the various causes in the different age groups. This frequency may well vary in different regions of the country, but, in our experience, the following classification seems acceptable.

INFANCY AND CHILDHOOD

Meningoencephalitis. In the very young child, meningoencephalitis is one of the most common causes of loss of consciousness. It may occur in the presence of an apparently mild systemic infection. The history will be helpful in revealing the presence of a febrile illness or an involvement of the middle ear or sinuses. The coma is often preceded by headache, malaise, nausea, or lethargy.

On examination, the temperature may be elevated and associated with a mild nuchal rigidity or even a skin rash. In the case of mumps, the presence of parotid swelling or exposure to the infection will be helpful. Diagnosis can be established by the presence of increased cells or organisms in the spinal fluid or by a positive blood culture.

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Treatment consists of specific chemotherapy directed toward the causative organism.

Postictal state. Coma appearing after a convulsion may be very difficult to evaluate, particularly if the physician is unaware of any previous seizures. This is often the case in the younger age groups when seizures first appear. If a history of previous convulsions is available, diagnosis is simplified. In the absence of such a history, the presence of frothing at the mouth or associated motor movements should suggest the correct diagnosis.

Examination often is not helpful. Scars on the tongue or bruises on the body may suggest the correct diagnosis. Laboratory studies offer little aid during the acute episode. If the possibility of a postictal coma is suspected, watchful waiting for a few hours often indicates the diagnosis. These patients invariably recover in a few hours without residual effects.

Treatment is not necessary during the coma. After recovery, adequate investigation as to cause and medicinal control of the seizures should be instituted.

Toxins. Occasionally, youngsters accidentally ingest hypnotic drugs which have been discarded or poorly concealed. A deep coma then develops without any history or findings of a systemic reaction. Examination usually reveals only suppression of all the deep reflexes. Laboratory studies are not helpful. If such a condition is suspected, the patient should be treated symptomatically, paying special attention to the vital signs. Recovery is usually spontaneous after a few days, without untoward complications.

Hypoglycemia. This type of coma is frequently seen in poorly regulated juvenile diabetics or in infants as a spontaneous episode. In the former, the history of diabetes immediately suggests the diagnosis. In spontaneous hypoglycemia of infants, the history indicates an episodic syndrome occurring before meals and characterized by restlessness, irritability, convulsions, and, finally, coma.

Examination often reveals increased sweating, rapid pulse, and motor restlessness. Diagnosis is established by the blood sugar level.

Treatment consists of proper administration of glucose. Infants with a metabolic disturbance can be regulated by corticotropins.

YOUTH

Postictal state. This is by far the most common cause of coma in this age group. The diagnosis has been discussed in the previous section.

Subarachnoid hemorrhage. If an adequate history of the onset of the illness can be obtained, the diagnosis is readily apparent. In an apparently healthy individual, a very acute, intense nuchal headache develops, which is associated with vertigo, vomiting, and lethargy, which soon deepens to coma. The diagnosis is not so readily apparent if the patient is found in a comatose state.

Examination often reveals two characteristic features: (1) the neck invariably is stiff and (2) occasionally, on ophthalmoscopic examination, subhyaloid or "sheet" hemorrhages are seen. The diagnosis can be verified by the presence of a bloody spinal fluid.

Treatment during the acute period is symptomatic. If the patient survives the hemorrhage, recovery ensues within days or weeks. After recovery, the patient should be investigated and treated for the causative factor, which is most often a ruptured aneurysm.

Head trauma. Without an adequate history, diagnosis can often prove very difficult. Many children, as well as young adults, receive head injuries while alone and become confused or lethargic before they can offer an adequate history.

In this age group, examination should include a careful search for fresh scalp or body bruises, bleeding from the nose or ears, or a fracture. A spinal puncture may be helpful by revealing a blood-tinged spinal fluid. A skull roentgenogram should always be carried out as a means of detecting fractures.

Treatment is symptomatic. Fortunately, most patients with milder head injuries recover without residual effects. In the more severe cases, some cerebral edema, with restlessness, irritability, and bradycardia may develop. In such cases, dehydration may be obtained by magnesium sulfate enemas or intravenous urea. If these procedures fail to relieve the pressure, surgical decompression may be considered.

Drug intoxication. This is a relatively common cause of coma in the young adult. Often the patient is discovered unconscious, and examination reveals no evidence of a systemic process and no neurologic abnormalities. If a history can be obtained, it often reveals some emotional

instability, depression, or insomnia, for which hypnotics have been used. Social or environmental conflicts often precipitate a suicidal attempt.

Examination is not helpful except for the suppression of the deep reflexes. Laboratory studies may be helpful in revealing elevated quantities of barbiturates in the urine or blood.

In milder cases, treatment is symptomatic. After a few days, the patient begins to recover spontaneously. In severe cases, the airway must be kept open by suction or even tracheotomy. The vital signs must be carefully watched. Caffeine sodium benzoate in intravenous doses of 0.5 gm. is often helpful. Analeptics should be used only if all responses to stimuli are absent. In such cases, picrotoxin administered intravenously at the rate of 0.5 cc. per minute up to 25 cc. may be used. With use of this drug, care should be taken to avoid convulsions. The drug should be discontinued as soon as the patient shows evidence of responding to stimuli.

Postictal state. Although not as common as in the younger age groups, coma following convulsions is occasionally seen in adults as a presenting illness. The findings and diagnosis have already been discussed.

Diabetic coma. Most patients with diabetes severe enough to produce coma are aware of their illness and are under treatment for it. Therefore, in most of these cases, a history of the illness is available. In the absence of such a history, diabetes should be suspected if there is a history of generalized weakness, polyuria, polydipsia, and polyphagia.

During coma, the findings are very suggestive. The patient appears dehydrated, with sunken eyes and soft eyeballs. The breathing is slow and labored (Kussmaul) and the breath smells "fruity" or of acetone. The pulse is rapid and feeble and the blood pressure low. The fundi occasionally reveal a typical diabetic retinopathy. Examination of the urine or the blood promptly verifies the diagnosis.

Treatment consists of restoring salt and water with 1,500 cc. of saline in order to arrest dehydration and correcting the glycosuria, ketosis, and acidosis by proper use of insulin.

Hysteria. If obtainable, the history may reveal the emotional instability that precipitates the episode. A patient in hysterical coma does not respond to the usual noxious stimuli. The neurologic examination is entirely negative. The diagnosis is suggested by the absence of any positive findings and by the resistance of the patient to usual examination procedures, such as opening the eyelids.

In this age group, by far the most frequent causes of coma are cerebral lesions. One of these that occurs most often is cerebrovascular disease, particularly in individuals with hypertension. Coma in cerebrovascular disease is usually due to a hemorrhage rather than to a thrombosis.

Cerebral hemorrhage. These patients are often known hypertensives who have had previous attacks of focal brain disturbance. Many have prodromal symptoms of headache, vertigo, or transient motor or visual disturbances. The coma usually occurs during periods of activity and is often preceded by intense headache.

Examination reveals focal damage to the brain, such as anisocoria with enlargement of the ipsilateral pupil, paresis of one side of the body, sagging of one side of the face, or conjugate deviation of the eyes and head. Ophthalmoscopic examination often shows the typical hypertensive vascular changes. Spinal puncture, in many cases, will reveal a blood-tinged spinal fluid, occasionally under increased pressure.

Treatment during the acute stage is symptomatic. If the patient survives, the outlook for functional recovery is good with the use of modern rehabilitation procedures.

Cerebral embolism. The history is often helpful by revealing previous cardiac disease, particularly fibrillation or myocardial infarction. If an adequate history is not available, the patient must be examined carefully for a fibrillating heart and the electrocardiogram must be used to aid in the establishment of a previous myocardial infarction. Neurologic examination almost invariably reveals evidence of bilateral or widespread cerebral involvement, which is so suggestive of emboli.

Treatment consists of anticoagulant therapy. With such therapy, the prognosis is excellent if the emboli are mural in origin.

Subdural hematoma. This diagnosis should always be suspected in the presence of a history of trauma or alcoholism. Generally, the coma is slow in onset and develops over a period of weeks. It is usually associated with some focal symptomatology and, in many cases, with papilledema.

The most important single laboratory study is a skull roentgenogram, which should be examined carefully for a shift in the position of the pineal gland. If the diagnosis is suspected, it can be verified by angiogram or air encephalogram.

Treatment consists of surgical removal of the lesion.

Brain tumor. On rare occasions, coma may be the presenting manifestation of tumors situated in the frontal or temporal regions. Usually, a careful history reveals preceding headaches, seizures, or slowly developing mental changes, such as memory impairment or personality alteration.

Examination is helpful by revealing mild focal changes and perhaps papilledema. A skull roentgenogram may show a calcified displaced pineal gland. Diagnosis is confirmed by angiography.

Alcohol. Alcoholic stupor is readily suspected by the history of chronic drinking. The condition can be established by the alcoholic odor of the breath. The urine or blood may be checked for its alcoholic level. Persistence of coma for more than a day should promptly suggest a diagnosis of some complicating process, such as a hematoma.

Uremia. The history is of help only if it reveals information regarding previous kidney disease. In many cases, such a history is not available.

On examination, the patient is generally restless and may manifest muscle twitchings and even convulsions. Peripheral edema is often present, and the breath has the characteristic urinous odor. The fundi generally show typical renal retinopathy.

Diagnosis is easily verified by laboratory studies. The urine contains casts and albumin, and the blood metabolites are increased.

Treatment is palliative.

Hepatic coma. In some cases, a history of previous hepatic difficulty, such as abdominal discomfort, ascites, or jaundice, is obtained. Recurrent episodes of coma should always suggest hepatic disease.

In most cases, examination establishes the diagnosis. These patients have edema of the extremities, a rapid pulse, and a subnormal temperature. Spider nevi are often present about the face and neck, and a bleeding tendency is apparent. The breath often has a "fishy odor" (fetor hepaticus). Laboratory studies are helpful by revealing abnormal liver function (cephalin-flocculation and serum globulin tests).

Shock (blood loss). Occasionally, patients in this age group suffer subtle blood loss, to which they pay little attention. This is particularly true of women who may have slight bleeding from the uterus or bowel without obtaining medical attention. There results a gradual drop in hemoglobin, with decreased oxygenation of the brain. In such cases, any associated vasomotor disturbance or stress situation may precipitate a sudden loss of consciousness. History

in such cases is not helpful, and diagnosis must depend upon the clinical findings.

The patient is pale, with cold, clammy skin. The pulse is rapid and thready, and the blood pressure is low. Diagnosis can be determined by blood studies.

Treatment consists of prompt transfusion and in some cases may be lifesaving. After improving the blood picture, the cause of the bleeding should be investigated.

OLD AGE

Most of the causes of coma in this age group are the same as those discussed in the previous section. By far the most frequent cause is cerebrovascular disease, particularly intracerebral hemorrhage or a large cerebral infarction due to thrombosis.

One additional condition warrants discussion, namely, the sudden onset of coma due to *heart failure*. In many of these patients, the history reveals evidence of cardiac disease either in the form of therapy or in the nature of such symptoms as edema, dyspnea, difficulty in ascending stairs, and so on.

Examination of the comatose patient reveals an enlarged heart, evidence of pulmonary congestion with rales and fluid, and cardiac irregularities. An electrocardiogram and a heart roent-

genogram may aid in the diagnosis. These patients respond promptly to therapy. In many, the coma clears up merely by placing the patient in a prone position. The use of cardiac stimulants to improve cardiac function also aids in improving the patient's condition.

DIAGNOSTIC LEADS IN COMA

Keeping in mind the characteristic features of the various causes of coma in the different age groups, a list has been made of a few helpful leads in directing the physician to the correct diagnosis in such cases.

1. Edema of the ankles—heart failure, uremia, and hepatic coma.

2. Breath odor—alcoholism (alcoholic), diabetic acidosis (fruity or acetonemic), uremia (uriferous), and hepatic coma (fishy).

3. Stiff neck—meningoencephalitis, subarachnoid bleeding, and trauma.

4. Subarachnoid blood—subarachnoid hemorrhage, trauma, intracerebral hemorrhage, and, occasionally, subdural hematoma.

5. Ophthalmoscopic changes—subarachnoid bleeding (subhyaloid hemorrhage), diabetic coma (diabetic retinopathy), cerebral hemorrhage (hypertensive changes), tumor (papilledema), subdural hematoma (papilledema), and uremia (albuminuric retinopathy).

THE BENEFITS OF oral neomycin for treatment of hepatic failure far outweigh the hazards, but renal function and auditory acuity should be watched closely for signs of systemic absorption. Care must be taken to avoid too high doses; up to 6 gm. may be given daily. In most instances, 3 or 4 gm. daily suffices.

Oral neomycin was administered to 27 patients with acute and chronic hepatic insufficiency in doses rarely exceeding 4 gm. daily. Serum samples were assayed during therapy to determine if the drug was absorbed from the gastrointestinal tract.

Circulating neomycin was found in 7 patients. Development of blood neomycin levels could not be correlated with gastrointestinal ulceration but was related to development of oliguria. In 1 patient without oliguria, permanent deafness resulted from the systemic absorption of neomycin. No proof could be obtained that enough neomycin was absorbed to produce renal damage beyond that usually associated with terminal hepatic failure.

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Gastrointestinal Polyps: Familial Patterns

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AS DEFINED BY Burt,¹ the term polyp is merely a gross anatomic description applied to any pedunculated structure protruding from a mucous membrane. Polyps may be of any cell type, but this paper will be concerned mainly with benign adenomatous polyps and will consist of a review of the known syndromes of polyps of the gastrointestinal tract which occur as a familial trait. The symptoms, signs, and management will generally apply to other cell types as well.

ESOPHAGUS, STOMACH, AND DUODENUM

There is no recognized familial syndrome of polyps of the esophagus, and polyps of any kind are rare. Boyd and Hill² found only 2 in 13,460 autopsies in 1954.

While polyps of the stomach are relatively common, no familial pattern has been established. The tendency toward malignant degeneration is similar to that of polyps of the colon; consequently, they have the same clinical significance.

In the duodenum, polyps occur with some frequency, but there is no familial pattern. Polyps and tumors of the duodenum tend to be malignant (15 out of 23 in one series³ were malignant) and often produce symptoms indistinguishable from peptic ulcers.⁴ Because of the relative immobility of the duodenum, intussusception is not a problem. However, obstruction may occur if the polyp becomes large enough.

SMALL BOWEL: PEUTZ-JEGHERS SYNDROME

Since the popularization of the syndrome of intestinal polyposis with mucocutaneous pigmentation by Jeghers and associates⁵ in 1949, diag-

nosis of adenomatous polyps of the small bowel without pigmentation is no longer fashionable and is not mentioned by contemporary authors. The 339 cases of multiple polyps of the small bowel collected by Shaw⁶ in 1939 are presumed to have been the modern syndrome, the characteristic pigmentation having been overlooked.

Peutz-Jeghers syndrome consists of: (1) multiple intestinal polyps, usually benign adenomas, occurring mainly in the jejunum and ileum; (2) a history of familial involvement; and (3) mucocutaneous melanin pigmentation, especially of the buccal mucosa.

History. The first case of what today would be called Peutz-Jeghers syndrome was described in 1881 by Skifasowski.⁷ In 1896, Hutchinson⁸ described buccal pigmentation in twin sisters, one of whom later died of intussusception at the age of 21. In 1921, Peutz⁹ first described a case in which he pointed out the association of polyps, pigmentation, and familial occurrence. However, his publications went largely unnoticed until Jeghers and associates,⁵ in 1949, reported 10 cases and redescribed the syndrome. In 1957, 60 cases were collected from the literature by Dormandy,¹⁰ and, since then, about 2 to 4 cases a month have been reported.

Familial aspects. Approximately 40 per cent of cases have a positive family history, and pedigrees suggest that a dominant gene, usually of high penetrance, is responsible. The association of dissimilar traits thought to be due to a single gene is not unusual in a syndrome, for example, polycystic kidneys and cerebral aneurysms, neurofibromatosis and café au lait spots, and so forth.¹⁰ Modern genetic theory discounts the existence of pleiotropism—the hypothesis that multiple primary actions may result from the presence of a single gene—and suggests that the gene for Peutz-Jeghers syndrome may act instead in one

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of a number of ways: (1) by activating a group of genes, (2) by lowering the threshold for phenotypic expression of various characteristics, or (3) by causing temporary but widespread imbalance at some embryonic stage of development of mucous membranes in general. This last theory is used to explain other multiple abnormalities that have been noted in some of these patients, such as bladder, nasal, and bronchial polyps and skeletal defects such as scoliosis, multiple exostoses, and fibromas. The occurrence of sporadic cases is attributed to spontaneous mutations since, in all other respects, such cases are identical with the familial cases.

Pigmentation. Clinically, the syndrome is characterized by mucocutaneous pigmentation and multiple intestinal polyps, with a familial tendency. The cutaneous pigment appears early in infancy or childhood and is located usually on or around the lips, occasionally on the soles of the feet and palms of the hands, and rarely elsewhere. This lesion takes the form of small, dark brown or black maculae, which often fade and may completely disappear at puberty. These blemishes are nonhairy, nonvascular, nonirritating lesions that do not rise above the surface of the skin and that have not been reported to undergo malignant degeneration. When they persist, a dermatologist or plastic surgeon may be consulted for cosmetic reasons.¹⁰

The mucosal pigment appears at an early age and is distinctive in that it persists throughout life. The spots consist of brown to black deposits of melanin, most commonly appearing on the buccal mucosa, gums, palate, and nasal lining but never in the conjunctiva, rectum, or intestine itself. Originally, it was thought that pigmentation occurred only in dark-complexioned individuals,⁵ but now it is recognized that it occurs with equal frequency in light-complexioned persons, where it contains less melanin and is harder to identify.¹⁰

In contrast, the pigmentation of freckles occurs chiefly on the cheeks and nose, is never present at birth, and is never seen on the buccal mucosa. Freckles are said not to occur in patients with Peutz-Jeghers pigmentation. The pigmentation of Addison's disease is very similar in its morphologic appearance but generally is accentuated in body folds and appears later in life. Similar pigmentation also has been reported in such conditions as pernicious anemia, sprue, and celiac disease.

Polyps. Polyposis is the main clinical feature and is responsible for the symptoms which usually bring the patient to the doctor. These polyps are usually most numerous in the small bowel,

particularly in the jejunum and ileum, although cases have been reported³ in which the entire gastrointestinal tract, from the cardia of the stomach to the rectum, has been involved. The polyps are usually multiple but seldom reach the massive numbers found in colonic polyposis.

Symptoms associated with these polyps are due to transient, self-reducing, often multiple and always recurring episodes of intussusception, giving rise to brief but severe bouts of abdominal colic. Patients often report loud borborygmi, arising from lumps that they can sense and occasionally palpate, "rolling about inside" during an attack. These usually end abruptly, often with the simultaneous passage of a good deal of flatus, in response to some movement, massage, self-induced emesis, or other trick that the patient has learned, only to recur again hours, days, or months later. Commonly, these attacks occur ten to fifteen minutes after a meal or upon arising in the morning and almost never while resting or at night. It is no wonder that the majority of these patients, if seen in the intervals between attacks, might be labeled neurotics, presenting as they do a history of inconstant, vague abdominal pain in different locations at different times, during which time the patient feels "noisy lumps rolling about." This history, in addition to good general health and the absence of abdominal findings between attacks, conspires against correct diagnosis of the patient.¹¹

Polyps in this condition, as is true of small bowel polyps and tumors in general, may also manifest themselves by the presence of an unexplained anemia and of occult blood in the stool. In patients with the findings of silent blood-loss anemia, the small bowel and cecum should be investigated. Small bowel fluoroscopy is imperative in these cases and may need to be repeated in order to demonstrate polyps. Attempts to pass a Miller-Abbott tube and obtain an air-contrast study of the small bowel also have been tried in an effort to demonstrate polyps.

Diagnosis of this syndrome usually depends on the patient's story because, in spite of its bizarre sound, it is remarkably consistent from person to person. The finding of the pigmentation and, perhaps, a positive family history, occasionally in association with anemia and occult blood in the stool, complete the picture.

The tendency to malignant degeneration in this syndrome is in dispute. Berkowitz and associates¹² feel that 25 per cent of the polyps will become malignant, but most writers¹³ believe that malignancy is rare. In support of this is the oft-quoted statement that there are no cases where

metastases or deaths from small bowel adenomatous polyps have been demonstrated. Dorman¹¹ says that adenomatous polyps probably arise from primitive adenomatous vesicles embedded in the bowel, and this probably accounts for the pathologic interpretation of infiltration by the polyp.

The generally accepted management of these patients, in view of the rare presence of malignancy, is conservative. In those instances where the patient's symptoms are short-lived and occur only infrequently, he should be informed as to the nature of the disease and followed with periodic hemoglobin and stool guaiac tests. In cases of persistent difficulty, exploration with polypectomy or resection of an affected segment should be advised. As much small bowel as possible should be salvaged, because new polyps may appear and repeated "nibbling" of sizable segments of bowel could conceivably result in malabsorption and other problems.

LARGE BOWEL: FAMILIAL POLYPOSIS OF THE COLON

Familial polyposis of the colon is a hereditary condition transmitted, like Peutz-Jeghers syndrome, by a dominant gene of high penetrance; one half of the offspring of a heterozygous parent will inherit the gene and exhibit the disease. Solitary cases may occur which appear and behave similarly to the familial ones.¹⁴

Dukes and Lockhart-Mummery,¹⁵ in their twenty-five-year study at St. Mark's Hospital, have followed 50 families with over 1,000 members, 208 of whom have polyps and 150 of whom have cancer.

The mean age for the appearance of symptoms is 21 years. Diarrhea and lower gastrointestinal bleeding are the common manifestations. Of 201 cases studied at the Mayo Clinic,¹⁶ 17 exhibited Gardner's syndrome exostoses, sebaceous cysts, or connective tissue neoplasms occurring in "association with this condition."

Diagnosis of the disease is best made by sigmoidoscopy, all cases ordinarily having polyps in the rectum. In suspected cases, a barium enema should be done as well. Sometimes, the polyps are so numerous that the physician cannot "see the trees for the forest," and the diagnosis may be missed. Ulcerative colitis may usually be distinguished by the granular and inflamed intervening mucosa.

The malignant potential of these polyps is well known and marked. It has been said that 50 per cent of these patients will harbor infiltrating colonic cancer by the age of 30, and virtually all will develop cancer by 50.

Management of this disease consists of diagnosis and treatment of the individual case and, perhaps even more important, long-term follow-up for cancer detection in other family members.¹⁷ Generally, prophylactic surgery can be deferred until the age of 25 in cases that are asymptomatic. Dukes and Lockhart-Mummery¹⁵ believe that colectomy with ileorectal anastomosis, fulguration of all rectal polyps, and proctoscopic examination every three months for five years and then every six to twelve months for life is the procedure of choice. Any new polyps should be fulgurated each time. Usually only a few appear, and sometimes none will develop after colectomy. These patients may expect some diarrhea for a few weeks but generally settle down to 2 to 3 formed bowel movements a day thereafter. However, in cases where malignancy already exists or in cases where there are so many rectal polyps that fulguration would lead to excessive scarring and subsequent stenosis, total proctocolectomy with permanent ileostomy should be advised.¹⁷

In other family members, sigmoidoscopy as described is sufficient to rule out the existence of significant polyps. Children of parents who display the condition need not be examined until the age of 13 unless symptoms supervene, as early polyps and malignancy are rare. Thereafter, examination every five years is recommended. Such children should receive an explanation of the situation, and the importance of periodic examination should be stressed at this first meeting. Children whose parents are unaffected or adults over 40 without polyps or symptoms need not be followed. The English authors¹⁵ stress the importance of a careful, long-term follow-up program to adequately care for these people and detect new cases.

SUMMARY

1. Polyps of the upper gastrointestinal tract are not known to occur in familial patterns.
2. Peutz-Jeghers syndrome consists of multiple intestinal polyps occurring mainly in the small bowel, mucocutaneous pigmentation, and a familial tendency.
3. The polyps give symptoms of recurrent paroxysmal episodes of intussusception and occasionally give rise to anemia and gastrointestinal bleeding.
4. These polyps tend to remain benign, and medical and surgical treatment is therefore expectant and conservative.
5. The buccal mucosa is the most constant site for pigmentation.
6. Familial polyposis of the colon is the com-

plementary condition in the large bowel, but, because of the marked tendency toward malignancy, prophylactic resection of the bowel at a precancerous stage is recommended where possible and long-term follow-up is mandatory.

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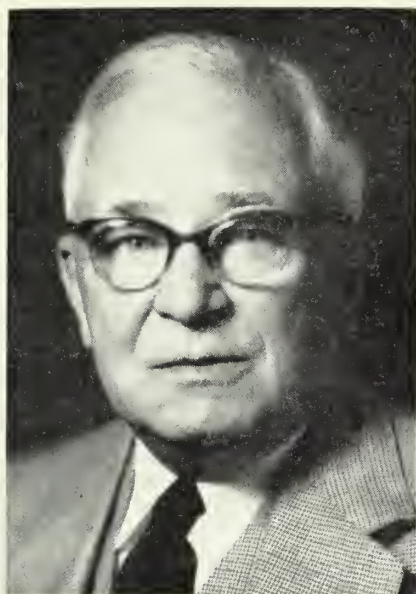
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SKIN REACTIONS TO 1 mg. of old tuberculin (OT) or to second-strength purified protein derivative (PPD), or 0.005 mg., are not the result of chemical irritation but are true allergic reactions. Lack of reaction to such doses in reasonably healthy persons practically eliminates the possibility of infection with acid-fast organisms or tubercle bacilli.

Persons reacting strongly to intermediate-strength PPD, or 0.0001 mg., or failing to react to second-strength PPD are less resistant to development of overt tuberculosis than healthy persons reacting only to second strength. The low-grade sensitivity assures relative immunity, whether induced by atypical acid-fast organisms, other unknown organisms, or true tubercle bacilli.

Of 120 patients with known tuberculosis, 30 per cent reacted to skin tests with OT in a 1:10,000 dilution. With 1:1,000 OT, the percentage of reactors rose to 69 per cent, and 85 per cent reacted to 1:100 OT. In tests with first-strength PPD, 84 per cent of 81 patients reacted; 5 per cent reacted to second strength, and 10 per cent to intermediate.

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Raymond G. Arveson, M.D.

1883–1960

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WHEN AN ENTIRE COMMUNITY honors a man with a public demonstration, it is usually for some outstanding achievement which has benefited that community. Dr. Raymond G. Arveson, a general practitioner in Frederic, Wisconsin, for fifty-two years, received such public recognition not once but twice in the five years preceeding his death on June 5, 1960.

In 1956 on the fiftieth anniversary of his becoming a physician, Dr. Arveson was honored by the Wisconsin State Medical Society. At that gathering, Dr. W. B. Stovall, professor of hygiene at the University of Wisconsin, said, "His leadership is inspiring, remarkable. He is a citizen as well as a doctor. He is everything a doctor should be. I hope the tradition he has set will live on with us in Wisconsin medicine." Dr. E. M. Dessloch, Prairie du Chien, Wisc., added, "Dr. Arveson has made our lives fuller, richer, and more meaningful. He is the man we love . . . a great man in medicine."

On Dr. Arveson's 75th birthday in 1958, his friends—including practically the entire population of Frederic and Polk County, plus many persons from all parts of the state—honored Dr. Arveson for his fifty years of loyal service as a Frederic physician and as a leader in civic, religious, and educational projects. The program started with a dinner attended by guests from many professions, and by physicians and their wives from across the state. The evening was climaxed by an open house in the Frederic High School gymnasium, where hundreds came to greet and honor "their doctor" and his wife.

Dr. Arveson was born at Duluth, Minnesota, in 1883, but soon moved to neighboring Wisconsin. The young physician who arrived in Frederic in 1909 had graduated from the Wisconsin College of Physicians and Surgeons, Milwaukee—a school which

merged with Marquette University in 1906. He had served a year's internship at St. Joseph's Hospital, Milwaukee, after which he had gone to Hiles, Wisconsin, as physician and surgeon for a lumber company. He then moved to Frederic.

Frederic was not much of a village when the aspiring young physician arrived. In fact, the village was only 8 years old when Dr. Arveson joined forces with its population of 450 persons. Frederic, which had originated with railroad and lumber activities, was on the downgrade. The lumber had been cut and a fire had reduced a good part of the main street to shambles. Even in that period of unattractiveness, there must have been something about Frederic and its people which attracted the doctor; something which was to keep him there for fifty-two years.

At first, the youthful physician did not plan to remain in Frederic. "During medical school we were always told to go out in the country and make some money, and then come back to Milwaukee to practice," he said. But Dr. Arveson had formed a philosophy for his life's work from which he was never swayed. "To me it has always been a duty to do the things you can do." And in Frederic there were many things he could do and did. He was one of those medical pioneers in the days of the horse and buggy doctor. Winters were rugged in those days. For many years his calls were made in a cutter. Bells saved from the shaft of his cutter became the nucleus of his collection of bells—a collection which now has bells sent from all parts of the world. The bells have been donated to the state medical society museum at Prairie du Chien.

Soon after his arrival in Frederic, Dr. Arveson married Miss Mabel Chandler of Eau Claire, Wisconsin. She proved to be a charming helpmate who

was always interested in his career as a physician. She, too, soon grew to love the community, and became involved in many civic and religious projects. The couple was saddened by the death of a daughter, Barbara Jean, in 1928.

Dr. Arveson's interest in the growing community was shown by his concern about everything affecting Frederic. Changes came after the horse and buggy days, and Dr. Arveson kept up not only with progress in medicine but also with standards of community welfare. When any change was suggested, Dr. Arveson always thought, "Is this change good for the community?" If convinced that it was, he worked tirelessly for the success of the idea.

One of the community's great needs in those early days was a hospital. So, in 1916, Dr. Arveson started the first hospital built in northern Wisconsin. That first hospital had a capacity of 6 beds. The Frederic Hospital today is a 30-bed institution, and one of the most modern in northern Wisconsin. In 1945, the hospital was turned over to the village of Frederic to be operated as a municipal hospital, but Dr. Arveson continued to serve as chairman of the board.

To advance better service for his patients and the community, he next established a clinic. In 1916, a schoolmate and close friend of Dr. Arveson's, Dr. John A. Diamond, came to Frederic. With him, Dr. Arveson founded the Arveson-Diamond Clinic. The partnership clinic existed until Dr. Diamond left Frederic in 1937. Dr. Walter C. Andrews, who came to Frederic in 1926, formed the Arveson-Andrews Clinic. In 1945, when Dr. Robert M. Moore and Dr. William A. Fischer became associates of Drs. Arveson and Andrews, the name was changed to the Frederic Clinic. The clinic is now housed in the municipal hospital building.

Dr. Arveson generously contributed his time and proved his ability through active participation and counseling on the many community projects which have made Frederic a thriving village. He served as village president at various times throughout a period of twenty-three years. His name appeared with any committee or group formed for the betterment of Frederic. Education of the young always greatly interested Dr. Arveson; he served for years on the local school board and as its supervisor.

He was always ready to prescribe for the town's ills as well as for those of its inhabitants. When a tragic fire in 1934 totally destroyed the old frame St. Luke's Methodist church, Dr. Arveson's enthusiasm spearheaded the almost immediate start of a new church. Dr. Arveson never settled for second best for his community. When Bishop Garth, the Methodist Bishop of Wisconsin, viewed the new church for the first time he called it "Wisconsin's little cathedral."

Throughout his life, Dr. Arveson was a religious leader. In an editorial tribute to him, the pastor of St. Luke's Methodist church, the Reverend L. Keith Hanley wrote, "Dr. Arveson's leadership throughout the years in the Frederic Methodist church has sel-

dom been equaled. It is the kind of intelligent vision that seeks to use the finest accomplishments of men in art, music, architecture and literature to the glory of God. His concern for the less tangible things of life has not kept him from going into his shop to build scenery for a Christmas play or to construct an ornamental lighting fixture or to don his old clothes to put creosote on a door. Nothing large or small has gone unnoticed that would make the church comfortable, attractive, and durable."

Pastors who served the church were always encouraged by Dr. Arveson. According to the Reverend Hanley, the doctor appreciated cultural and scholastic attainments; but most of all, he possessed a sense of compassion and the courage to stand by his convictions. "His leadership and interest have attracted many other fine men into the life of the church." The Reverend Hanley mentioned that whenever he attended out-of-town ministerial meetings, former pastors and friends of the doctor always asked "How are Dr. and Mabel Arveson?" His wife, for years a Sunday school superintendent, has been as diligent a worker for the church and the community as her husband.

Dr. Arveson's accomplishments were not limited to his own community. He was a member of the Board of Regents of the University of Wisconsin for eight and one-half years. At the time Dr. Arveson was on the board, he was the boss of Dr. Edwin B. Fred, now president emeritus of the University of Wisconsin. At the ceremonies observing Dr. Arveson's fiftieth year in Frederic, Dr. Fred had this to say of his long-time friend, "Never have I known such a man. He is a sincere friend of all the people." He told of Dr. Arveson's great gift for making people feel at ease and of his excellent sense of humor. It was also at this gathering that Charles Gelatt of La Crosse, Wisconsin, a member and former president of the Board of Regents of the University of Wisconsin, told of first meeting Dr. Arveson on the board. He explained how at ease he felt among the older members, because Dr. Arveson "made it easy for all of us to do our best."

Dr. Arveson's dedicated work through the years enriched the knowledge of the medical profession, as did the efforts of many pioneer practitioners. The Polk County Medical Society owes its existence to Dr. Arveson, who promoted its organization and first meeting in 1930.

His long years of experience, his profound knowledge of medicine, and his contributions to the ethics of his profession were known far beyond the borders of Frederic or Polk County. In 1940, he was paid the highest honor which can come to a physician. He was elected president of the Wisconsin State Medical Society. He served on the Wisconsin State Board of Examiners from 1941 to 1948 and was a member of the medical council from 1942 until his death. He was active in many organizations, including the Polk County Medical Society, the American College of Surgeons, and the Interstate Postgraduate Medical Association of North America.

Doctor Arveson's home has been in a small place, but he has never been isolated. Soon after the turn of the century, medical discoveries were made in every direction, and Dr. Arveson always kept pace with the rapidly advancing trends. His only lengthy absence occurred during World War II, when he served for a year in the Navy Medical Corps. Dr. Arveson never regretted remaining in a small town. He said on his 75th birthday, "There is no question but that one who works in a rural community can more readily find human and spiritual values than is likely for one who practices in a big city."

At the community celebration in his honor, Dr. Arveson reminisced on the fifty years since his arrival in the village. Humbleness was one of the doctor's greatest virtues. After being praised for his service to his patients and community, he said "One man doesn't make a village or a community. He may set the example, but it takes the work of everyone to see its fulfillment. Two women have been greatly responsible for my accomplishments—my mother and my wife. My wife and I could never leave here now—everything we have is here. We have greatly enjoyed it all, although the fifty years have gone by very fast, and many of the events I can recall seem to have happened only yesterday."

He may have been modest about his own accomplishments, but he was very ready to give credit to others. "Thousands of doctors in the United States have done a lot for small towns. I am only one. Ministers, priests, educators, professional men, and others form a nucleus of service to their communities." Of his own career, Dr. Arveson only said, "I have worked all the time—worked, worked, worked, and worked. That is the story of my life." And this, he said, had kept him happy.

But the many friends of Dr. Arveson are willing to talk on any occasion of his achievements. A bronze plaque, presented by friends at his fiftieth anniversary celebration, is now installed at the entrance of the Frederic Municipal Hospital. The plaque reads: "We honor Dr. Raymond Gregory Arveson, physician and surgeon. Founder of the Frederic hospital, president of the village of Frederic, president of the State Medical Society of Wisconsin and chairman of its council. Regent of the University of Wisconsin. Fifty years of service to his state, his community, and his fellowman. The final test of a leader is that he leaves behind him in other men the conviction and the will to carry on."

This sketch was written prior to the death of Dr. Arveson who died on June 5, 1960.

SLIGHT ATRIOVENTRICULAR block or intermittent disturbance with partial or complete block often disappears during treatment with prednisone. The P-R interval usually is reduced rapidly. Therefore, it is superfluous to continue treatment if improvement is not notable in four or five days. In 7 of 10 patients with slight block, 2 with partial block, and 5 of 7 with complete but intermittent block, daily doses of about 40 mg. of prednisone restored normal cardiac rhythm. The failures were all in patients with diabetes or receiving digitalis therapy. Ventricular rate was moderately increased in 12 patients with complete, permanent block, but no other electrocardiographic improvement was seen.

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A Clinical Pharmacologic Evaluation of Diethylpropion

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OBESITY is a common defect of a great proportion of the American population. Predominantly, obesity, or overweight, is the result of loss of balance between food consumed and energy expended, the balance becoming positive. Excessive food consumption for the overweight person frequently is, or becomes, a habit and is difficult to overcome.

Methods to control appetite encompass the entire field of both psychologic and drug therapy. Many persons with excessive appetites require a combination of both methods. However, until recently, most drugs have been ineffective in safe dosage as anorexigenic agents.

This report includes the results of a study of safety in small and large doses of a new drug in the field of appetite control.

Diethylpropion (Tennate) is a new anorexic which satisfactorily controls appetite and produces few, if any, of the undesirable side effects usually associated with such compounds. This compound differs chemically from the commonly used anorexigenic agents. Its structural formula is shown in figure 1.

Martin,¹ reporting the pharmacology, concluded: "Though the major pharmacologic activity of diethylpropion is apparently that of an anorexic, it is of great importance to emphasize the fact that this compound is comparatively lacking in the undesirable central nervous system stimulating effects found in similar compounds and that there are no observable toxic effects on the heart." The states of restlessness, nervousness, and inability to relax, which are typically caused by amphetamine, are not observed with comparable doses of diethylpropion.

In clinical trials, Spielman² observed that 25

mg. of diethylpropion given three to four times daily by mouth appeared to fulfill the criteria set up by Gadek and associates³ for the ideal drug in treating patients with the overeating syndrome. The drug appeared safe. It did not disturb the emotional and psychic balance; it did not require the concomitant use of barbiturates to counteract excessive stimulation; and it could be used in the evening without interfering with the patient's sleep.

Illig and Illig⁴ studied 48 diabetic patients who showed no blood glucose changes while taking the recommended dose of 25 mg. of diethylpropion orally three times daily.

Similar evidence showing that 25 mg. of diethylpropion administered orally three times daily is highly effective in suppressing appetite and reducing weight in obese patients, including cardiac, hypertensive, and diabetic patients, has been reported by Huels⁵ and Ravetz.⁶

The present study was designed to record the effects of the intravenous administration of diethylpropion in amounts equivalent to the recommended oral dose and also to 2 and 4 times the recommended dose. Data gathered by Martin¹ on the LD₅₀ for rats indicate that the equivalent intravenous dose of diethylpropion is one-tenth the oral dose. Therefore, the intravenous equivalent of the recommended single dose of 25 mg. was considered to be 2.5 mg. Twice the recommended oral dose would be 5 mg. and 4 times, 10 mg.

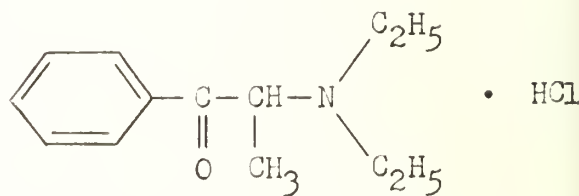


Fig. 1. Structural formula of diethylpropion HCl (Tennate).

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The diagnosis of the first patient in the series (table 1) was arteriosclerotic heart disease (A.S.H.D.) with mild hypertension and early heart failure. This patient was given 2.5 mg. of diethylpropion intravenously. Electrocardiograms were taken at rest before the injection and three and five minutes after the injection. Blood pressure, pulse, and respiration were taken at one-minute intervals for five minutes before and one-half, one, one and one-half, two, three, four, and five minutes after injection. No significant changes in the foregoing functions occurred. After a rest period of three days, the patient received 5 mg.; and, following another three-day interval, he received 10 mg. intravenously. Again, no significant changes were recorded (figure 2). Four days later, an electroencephalographic trac-

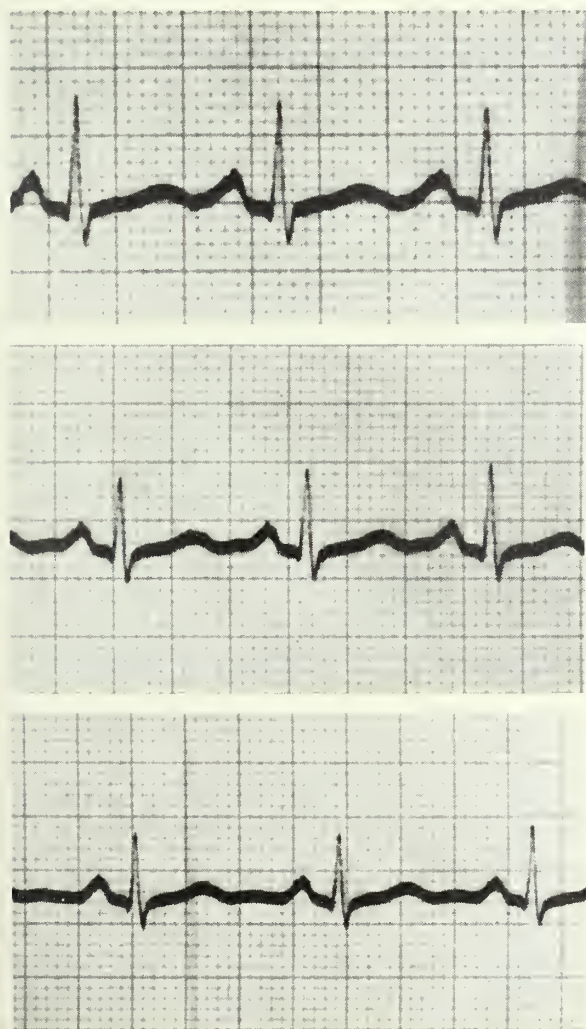


Fig. 2. Electrocardiograms (lead II) of first patient taken (*top*) at rest prior to intravenous injection of 10 mg. of Tenuate, (*center*) one and one-half minutes after injection, and (*bottom*) five minutes after injection.

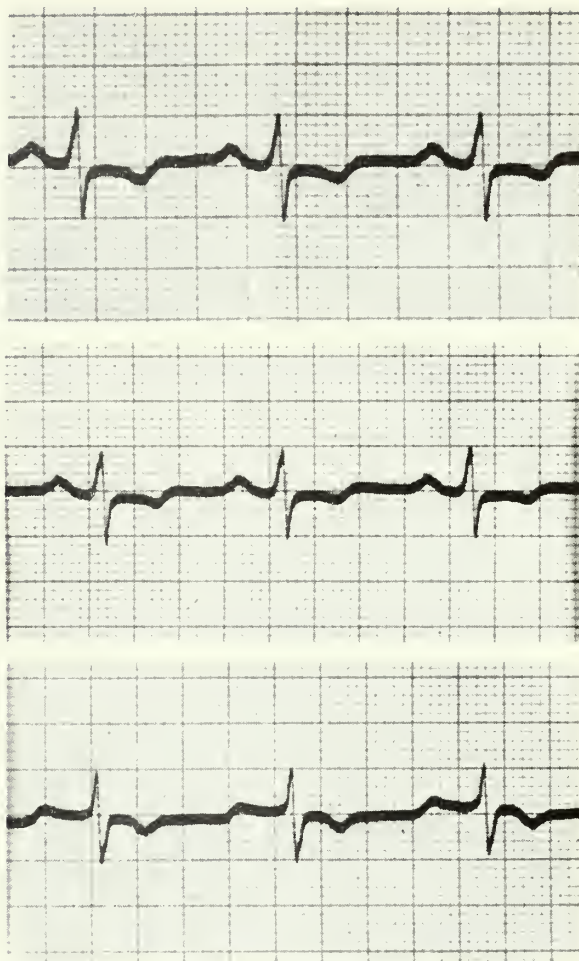


Fig. 3. Electrocardiograms (lead II) of second patient taken (*top*) at rest prior to intravenous injection of 10 mg. of Tenuate, (*center*) one and one-half minutes after injection, and (*bottom*) five minutes after injection.

ing was made. The patient was then given 10 mg. of diethylpropion intravenously and a second tracing was made. A prominent pulse artifact, which had been present previously, was greatly diminished in intensity after the drug was administered; otherwise, the second tracing showed no change from the first.

The second patient, with a diagnosis of compensated arteriosclerotic heart disease, mild diabetes mellitus, and postmenopausal obesity, was given 5 and 10 mg. intravenously at three-day intervals. Determinations were made before and after injection as described for the first patient. No significant alteration in the blood pressure, pulse, or respiration was observed. No electrocardiographic changes (lead II) were recorded after the intravenous administration (figure 3). The patients did not report any subjective complaints.

TABLE 1

EVALUATION OF INTRAVENOUS INJECTION OF 10 MG. DIETHYLPROPION (TENUATE)
IN PATIENTS WITH CARDIOVASCULAR DISEASE AND IN NORMAL SUBJECTS

Patient	Sex	Age	Diagnosis	Blood Pressure*		Pulse*		Respiration*		E.K.G. Tracing	
				Before	After	Before	After	Before	After	Control	After Injection
F.S.	M	67	A.S.H.D., mild hypertension; early congestive cardiac failure	175/90	175/90	81	81	17	18	Sinus rhythm; early LVH	No change
E.M.	F	62	Diabetes mellitus; A.S.H.D., compensated; obesity, postmenopausal	125/54	116/52	72	68	19	17	Sinus rhythm; non-specific myocardial changes	No change
M.A.	F	68	Essential hypertension; degenerative osteoarthritis; obesity	207/104	194/98	74	73	17	17	Sinus rhythm; normal tracing	No change
S.G.	M	30	Normal subject	126/74	122/78	86	82	17	17	Sinus rhythm; normal tracing	No change
D.A.	M	33	Normal subject	112/80	121/90	74	75	16	17	Sinus rhythm; normal tracing	No change
D.C.	M	55	Old antero-septal myocardial infarction; hypertensive A.S.H.D.; benign prostatic hypertrophy	136/95	125/93	76	74	16	16	Old antero-septal myocardial infarction; frequent V.P.C.s; non-specific S-T wave changes	No change
R.W.	M	62	A.S.H.D. with coronary insufficiency; angina pectoris; old posterior-myocardial infarction	142/90	138/90	96	95	19	18	Old posterior myocardial infarction with persistent ischemic changes; digitalis effects	No change
B.E.	M	68	A.S.H.D. with coronary insufficiency and angina pectoris; old posterior wall myocardial infarction; hypertension	174/94	168/91	54	54	22	21	Myocardial infarction; peri-infarction block; digitalis effects	No change
F.M.	M	70	Hypertensive A.S.H.D., compensated; chronic bronchial asthma with pulmonary emphysema; possible old antero-septal myocardial infarction; angina pectoris	133/52	141/50	60	57	24	25	Myocardial infarction; slightly prolonged inter-ventricular conduction; digitalis effects	No change
A.K.	M	62	A.S.H.D. with coronary insufficiency; angina pectoris; chronic cholecystitis; benign prostatic hypertrophy; chronic anxiety neurosis	145/89	145/91	78	76	16	17	Minimal S-T changes	No change
C.W.	M	73	Hypertensive A.S.H.D.	168/79	189/82†	72	70	18	18	Sinus rhythm; left ventricular hypertrophy; digitalis effects	No change
G.M.	F	57	Hypertensive A.S.H.D.; generalized and focal cerebrovascular pathology; left hemiparesis (as sequelae to O.V.A.); coronary heart disease with angina pectoris	233/110	243/122	63	66	20	21	Left ventricular hypertrophy; possible anterior wall myocardial ischemia	No change
J.B.	M	54	A.S.H.D.; mild hypertension; old posterior myocardial infarction; coronary insufficiency with anginal episodes; peripheral vascular insufficiency with intermittent claudication	162/100	160/103	92	91	19	19	Marked left ventricular hypertrophy; posterior wall ischemia	No change
L.V.	M	57	A.S.H.D. with coronary insufficiency; old anterior septal myocardial infarction	119/90	130/92	90	92	18	17	Old antero-septal myocardial infarction; non-specific S-T changes	No change
C.R.	M	39	Occasional V.P.C.s; nervous	186/116	186/117	85	86	20	20	Old anterior wall myocardial infarction; sinus rhythm with occasional V.P.C.s	No change

*These figures represent the averages of readings taken at 1-minute intervals for 5 minutes prior to injection and at $\frac{1}{2}$, 1, 1 $\frac{1}{2}$, 2, 3, 4 and 5 minutes after injection. Electrocardiograms for control were taken at rest before injection and at 3 and 5 minutes after injection. The amount of fluctuation between individual readings was considered insignificant.

†Since this rise of 23 mm. systolic was the only change suggesting any potential effect of diethylpropion on cardiovascular function in the entire series, the patient (C.W.) was retested 60 days later, using normal saline as the placebo. Prior to injection, his arterial pressure varied during a 5-minute period from 194/78 to 210/82. The increase produced by the intravenous placebo was 16 mm. systolic and 10 mm. diastolic. It was concluded, therefore, that in this patient any change following injection of diethylpropion or normal saline was caused by the psychic trauma associated with venipuncture rather than by either of the parenteral solutions.

A 68-year-old woman, who for years had suffered from degenerative osteoarthritis aggravated by obesity and essential hypertension, was chosen as the third patient. Her average blood pressure was 207/104; pulse, 74 per minute; and respiration, 17 per minute. She was given 5 mg. of diethylpropion after a thirty-minute rest period. Except for a modest drop in systolic pressure (13 mm. mercury in five minutes), no changes occurred in blood pressure, pulse, and respiration, or on the electrocardiogram (lead II). The test was repeated several days later using a 10 mg. dose. Findings were virtually identical (figure 4).

The next 2 patients were 30- and 33-year-old men whose case histories were negative for cardiovascular disease. Examinations revealed well-nourished and well-developed young men with no respiratory or cardiovascular pathology.

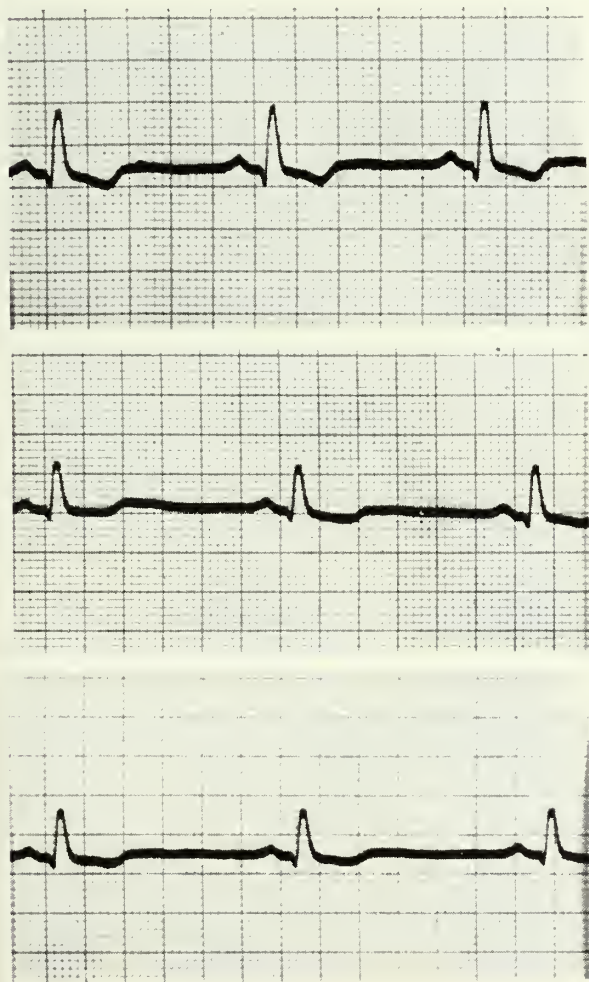


Fig. 4. Electrocardiograms (lead II) of third patient taken (*top*) at rest prior to intravenous injection of 10 mg. of Tenuate, (*center*) one and one-half minutes after injection, and (*bottom*) five minutes after injection.

Both were considered normal subjects and were given 5 and 10 mg. of diethylpropion on 2 well-separated occasions without significant change in any of the criteria, including the electrocardiogram (lead II).

At this time, we decided to give the drug intravenously to a group of 10 cardiovascular patients ranging in age from 39 to 73 years. Two patients in this group had hypertensive arteriosclerotic heart disease. One of them was a man who had suffered a cerebrovascular accident in 1958. At the time of examination, his blood pressure was 210/100. In addition, he had a digitalis-compensated, mild, congestive cardiac failure. The other patient was a woman who had suffered 4 cerebrovascular accidents with residual left hemiparesis. Her blood pressure upon examination was 232/120. Her electrocardiogram also revealed left ventricular hypertrophy, but there was no clinical decompensation.

The other 8 patients had coronary atherosclerosis. Seven of them had histories and electrocardiographic evidence of old myocardial infarctions of 7 months' to 5 years' duration. The remaining patient had a history of frequent anginal attacks, which have been relieved by nitroglycerin. His electrocardiogram revealed only minimal S-T changes. Of the 8 patients, 4 had associated hypertension and 7 had occasional to frequent anginal episodes and were currently taking nitroglycerin and other coronary vasodilators.

The results of intravenous administration of diethylpropion to this series of 15 patients are reported in table 1. They indicate that even in doses equivalent to 4 times the recommended oral dose, intravenous diethylpropion has no acute deleterious effect upon cardiovascular function, even in patients with heart disease. Accordingly, it would seem that this compound should be given serious consideration as a dietary adjunct when treating obesity complicated by cardiovascular disease.

CONCLUSIONS

A series of 15 patients, comprised of 2 normal subjects and 13 with diagnoses of essential hypertension, early congestive cardiac failure, post-cerebrovascular accident, coronary artery disease, and angina pectoris, were given doses up to 10 mg. of diethylpropion (Tenuate) intravenously. A dose of 10 mg. of diethylpropion is equivalent to 4 times the recommended single oral dose as determined by LD₅₀ studies in rats. Blood pressure, pulse, and respiration were taken at

one-minute intervals for five minutes prior to injection and at one-half, one, one and one-half, two, three, four, and five minutes after injection of the drug. Electrocardiograms were taken at rest before the injection and three and five minutes after injection. No acute deleterious effects of the drug upon blood pressure, pulse, respiration, or electrocardiograms were noted. An electroencephalographic tracing was made before and after the 10 mg. injection in 1 patient and each time was within normal limits.

The diethylpropion used in this study is available as Tenuate from the Wm. S. Merrell Company, Cincinnati, Ohio, and as Tepanil from the National Drug Company, Philadelphia, Pennsylvania.

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SHAM OPERATIONS AFFORD the same amount of subjective improvement in patients with angina pectoris as does internal mammary artery ligation. A patient's opinion of benefit from surgical or medical therapy for angina pectoris is an unreliable index of the effectiveness of the treatment. When a patient is told he has coronary artery disease, emotional factors become important in determining the intensity of symptoms. However, electrocardiographic changes with exercise are a constant and reproducible index of coronary insufficiency and are not necessarily related to the degree of the patient's symptoms, such as chest pain and exercise intolerance. Later terminal dipping of the RS-T segment of the electrocardiographic tracing is the most reliable indication of coronary artery disease.

Patients with angina pectoris had exercise electrocardiograms made by 2 cardiologists to establish definite proof of preoperative coronary artery insufficiency and to obtain objective evidence of the efficacy of surgical intervention. In 13 patients, the internal mammary arteries and veins were ligated. In 5 patients, selected at random by the surgeon without the knowledge of the cardiologists, the internal mammary vessels were exposed but not ligated.

Postoperatively, 15 of the 18 patients, including the 5 who had had sham operations, had definite and sustained relief from angina pectoris. Of the 15 improved patients, 13 had abnormal exercise electrocardiograms postoperatively, with changes comparable to those before operation.

E. G. DIMOND, C. F. KITTLE, and J. E. CROCKETT: Comparison of internal mammary artery ligation and sham operation for angina pectoris. *Am. J. Cardiol.* 5:483-486, 1960.

Transactions of the North Dakota State Medical Association

SEVENTY-THIRD ANNUAL MEETING

Grand Forks, North Dakota, April 30, May 1, 2, and 3, 1960

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7. Committee on Credentials:

R. E. MAHOWALD, Chairman
(also serves on Committee No. 6)
W. L. MACAULAY, Co-chairman
(also serves on Committee No. 4)

Grand Forks
 Fargo

PROCEEDINGS

Of the House of Delegates

Of the North Dakota State Medical Association

Seventy-Third Annual Meeting

The first session of the House of Delegates of the North Dakota State Medical Association was called to order by the Speaker of the House, Doctor G. A. Dodds, at 4:00 P.M. in the Dacotah Hotel, Grand Forks, April 30, 1960

Doctor R. E. Mahowald, chairman of the Credentials Committee reported that there was a quorum present and all credentials were in order.

Secretary Naegeli called the roll. The following delegates and alternates were present:

Drs. L. G. Pray, Fargo; F. A. DeCesare, Fargo; E. J. Beithon, Wahpeton; W. L. Macaulay, Fargo; J. B. Murray, alternate, Fargo; D. W. Palmer, Cando; J. Mahoney, Devils Lake; F. A. Hill, Grand Forks; W. C. Dailey, Grand Forks; R. E. Mahowald, Grand Forks; R. C. Painter, Grand Forks; W. W. Frey, Drayton; F. D. Naegeli, Minot; A. F. Hammargren, Harvey; R. Hordinsky, Drake; M. W. Garrison, Minot; C. J. Klein, alternate, Valley City; M. A. K. Lommen, Bismarck; M. Nugent, Bismarck; R. R. Tudor, Bismarck; C. Baumgartner, Bismarck; E. Vinje, Hazen; J. N. Elsworth, Jamestown; J. M. Van der Linde, Jamestown; D. R. Strinden, Williston; R. E. Hankins, alternate, Mott; and J. Little, alternate, Mayville.

The following also attended the House of Delegates meeting:

Drs. R. W. Rodgers, A. R. Gilsdorf, J. C. Fawcett, W. E. G. Lancaster, R. H. Waldschmidt, L. W. Larson, J. D. Craven, J. Sorkness, W. A. Wright, C. M. Lund, G. W. Toomey, C. J. Glaspel, O. A. Sedlak, C. H. Peters, T. E. Pederson, R. D. Nierling, G. Christianson, K. G. Vandergon, and Mr. Lyle Limond.

The motion was made, seconded, and passed that the reading of the minutes be dispensed with and that they be accepted as printed in THE JOURNAL-LANCET.

Motion was made by Dr. Baumgartner and seconded by Dr. Hill that Dr. Hanewald be elected as alternate delegate for the Ninth District in the absence of delegates Gilliland and Foster. Motion was passed.

Motion was made, seconded, and passed that the reading of the reports as printed in the Handbooks be referred to the proper reference committees and that the reading of these reports be dispensed with.

Secretary Naegeli next presented the following communications which were transmitted to the House of Delegates, addressed to the President, J. C. Fawcett, M.D.

Telegram to Dr. John Fawcett, president, and the House of Delegates and the Council of the North Dakota State Medical Association on the occasion of its annual convention.

I wish to thank the North Dakota State Medical Association and its members for the cooperation, the assistance, and the advice and counsel that have been given me ever since I became governor. All of us are aware today of the problem of providing adequate medical care for our senior citizens, a problem that must be solved. This solution must provide medical care for all who need it and cannot pay their own way in whole, or in part. I cannot support the so-called Forand bill because of its many and obvious shortcomings. This is a problem that involves the entire nation. It must be solved on a sound basis as promptly as all pertinent facts are available. I am hopeful that the work of the State and National Conferences on the Aging will give us a better understanding of our needs in this vitally important field. May I extend to you my most sincere wishes for a successful convention.

(Signed) John E. Davis, Governor

Letter from F. J. L. Blasingame, M.D., executive vice-president of the American Medical Association, Chicago, to J. C. Fawcett, M.D., president, North Dakota State Medical Association.

On the occasion of the Seventy-Third Annual Meeting of the North Dakota State Medical Association, I want to take this opportunity to express the sincere appreciation of the staff of the American Medical Association for the splendid program which has been carried out in the past few months in the field of national legislation.

In the current struggle to forestall congressional action in the field of compulsory health insurance, each state medical association in the country has faced a tremendous challenge. We feel that the North Dakota State Medical Association has responded to this challenge in a magnificent manner.

I hope that, on the occasion of your annual meeting, you will express our feelings to your officers, the councillors, the officers and members of your Committee on Legislation, the staff of your association, your congressional delegation in Washington, and, if possible, the officers of the county societies for their splendid cooperation in presenting medicine's views on the problems of the aging.

Also, please accept our best wishes for a most successful annual meeting.

(Signed) F. J. L. Blasingame, M.D.

Letter from C. P. Dahl, lieutenant governor, State of North Dakota, Cooperstown, to Dr. J. C. Fawcett, president, North Dakota State Medical Association.

It is with utmost sincerity that I extend my congratulations to the physicians of North Dakota during their annual convention for their untiring and successful efforts in providing highest quality care for the medical needs of the citizens of our State.

Since I have a son who is actively engaged in the practice of medicine in North Dakota, I am quite aware of the many personal sacrifices a physician must make so that these services are available to everyone at all times.

It is my contention that this quality service, which aims to overcome the physical and mental ills of mankind, can best be effected by sustaining the individual's prerogative of consulting the physician of his choice.

The practice of medicine must continue to be based on the principle of free enterprise, not subject to a social or government-regimented system.

A dilemma of growing national concern is that of providing care for the "medically indigent" of our aging American population. This problem must be met squarely and rationally by our elected representatives. The medical profession and every individual interested in this country's welfare must realize that this country became, and is, great because the individual's right has been protected under the law. I feel that financial support for any such program should be on the basis of need and not automatically include all persons of any particular age group regardless of personal solvency.

Any attempt to unadvisedly inject socialism into the practice of medicine in North Dakota or America must be defeated. If not, we will be well on our way into a welfare state or socialism which will eventually culminate in world communism.

(Signed) C. P. Dahl, Lieutenant Governor
State of North Dakota.

REPORT OF THE PRESIDENT

Twelve months ago, when I took over from Dr. Sedlak as president, a year seemed a long time. I can assure you that the time was too short for all the meetings, activities, and so forth developing during that time.

During the last annual meeting in Bismarck, we were confronted with complete disruption of the medical organization at the State Hospital in Jamestown. We offered to be of assistance in obtaining and securing new psychiatrists and staff, which offer was accepted (*apparently* with gratitude) by the governor and the chairman of the State Board of Administration. An advisory committee was appointed with Dr. Joseph Sorkness as chairman, who reports to me that Mr. Joos, chairman of the board, has never seen fit to use or consult the committee. I understand that replacements are pretty well completed, but that (1) qualifications of the new staff were never screened by our committee, and (2) a nonmedical superintendent still heads the institution.

During the year your Committee on Medical Economics had many meetings with the State Public Welfare Board, some of which I was privileged to attend. The results, I'm sure, will be covered in full in Dr. Peters' report. However, in the course of these negotiations, some ill-advised press releases came out from the Welfare Board office, such releases tending to put medicine in a "bad light" with the public. As a result of the completion of negotiations, the Welfare Board now has an advisory committee from the State Medical Association, on which is 1 physician from each geographic area of the state. It is to be hoped that better use will be made of this committee than was made of the Liaison Committee to the State Board of Administration.

If such is done, I would predict our relationship with the Welfare Board will be much happier in the future, and future negotiations will be much easier.

Blue Shield has agreed to adopt the Relative Value Fee Schedule (again see Dr. Peters' report), but action was delayed because of the trouble Blue Cross was having in effecting a rate increase, this being at least temporarily blocked by Mr. A. J. Jensen, our state insurance commissioner. In the course of discussions, Mr. Jensen released some comments to the press which were very derogatory to medicine, very ill advised, and contradictory to mandates he had previously issued to Blue Cross and Blue Shield. He even went so far as to comment on doctors' incomes, size of the cars they drove, and the kind of fur coats their wives bought. This, gentlemen, from one of the elective officers of our state!

From my report to this point, there can be but one conclusion—we have been missing the boat very badly, both in the area of our public relations activities and in our *collective* and individual participation in political and legislative action. We've not been winning the race, but it's not too late to get going. This means work not only by officers and committees of the association but active participation and work by the individual members of the society.

You are all well aware by now of the tremendous pressures in Washington by certain individuals and groups to push through Congress various measures to which we, as medical men, are irrevocably opposed, such as Forand-type legislation and compulsory social security inclusion of physicians. The Washington and Chicago offices of the AMA have become acutely aware that this threat cannot be licked at the national level but must be attacked at the grass roots level where the votes originate. Some of us attended an AMA sponsored legislative conference in

St. Louis in September where this fact was forcibly brought home to us.

We were fortunate that this was not a legislative year in North Dakota, but this coming winter, when the legislature will meet, there may be more legislation than usual initiated that will be detrimental to the best interests of medicine. The Committee on Legislation, with Drs. O. W. Johnson and C. H. Peters as co-chairmen, have been busy getting organized on both state and county levels, so that immediate pressures may be exerted on our representatives in Congress against this federal legislation. At the same time, *it is vital that we may have a complete state-wide organization* ready to go to work lobbying and contacting representatives and senators in our state legislature this coming January.

Considerable time was spent at the interim meeting of the council in December with regard to medicine's poor public relations and what to do about it. I'm sure it will again be on the agenda at this annual meeting and well it should be. I am firmly convinced that this is just as vital, or more so, than our participation in politics. *Actually the two are inseparable.* Sooner or later, I feel that we must employ a full-time public relations man. This will cost money, and the money must come from our dues. *If the dues must be raised to effect this, then by all means they must be raised.* It should have careful consideration, remembering that we are fighting on all fronts now, and the materials for fighting are not free.

It was my privilege to meet with several of the committees during the year. Particularly deserving of special mention for their dedicated efforts are Dr. C. H. Peters and his Committee on Medical Economics and Drs. O. W. Johnson and C. H. Peters and their Committee on Legislation. They have expended a tremendous amount of their time and efforts, as well as their own money. This is not to detract in any way from the thanks due to other committee chairmen and members that met and worked on problems during the year.

I attended the AMA meeting in Atlantic City in June. I have nothing to report that you don't already know of, except to note that it would appear that the chances of Dr. Leonard Larson to be elected president-elect in the coming year appear excellent. Also, our delegate, Dr. Willard Wright, was named to the important Council on Medical Service. One could not help but note the high esteem in which both Drs. Larson and Wright are held in the AMA.

Mr. Limond and I attended the meeting of the House of Delegates, South Dakota State Medical Association, in Rapid City in June. At that time, we officially invited their society to participate with us in our seventy-fifth anniversary meeting in 1962. The invitation has been accepted.

The Public Relations meeting in Chicago and the North Central Conference meeting in Minneapolis were very worthwhile and instructive. I can heartily recommend both of these meetings to any physician as well worth attending.

Dr. Larry Pray was our representative appointed by Governor Davis to the Washington Conference on Children and Youth. About the middle of October, the AMA sponsored a Regional Conference on Aging in Minneapolis, in which several of us participated or attended. It brought out the thinking as well as the ideas on this problem by various segments of our population: clergy, politicians, labor leaders, industrialists, and so forth. We will hear much more along these lines in the next few months. At the time of writing of this report, Governor

Davis is organizing a State Conference on Aging, in which conference our society anticipates having good representation and which should be held about the middle or latter part of March. This will be followed by regional conferences throughout the state.

I enjoyed very much visiting the various district societies. I was able to visit each society except the Northwest District, which a conflict in dates prevented me from visiting. I was represented there by your president-elect, Dr. Lund. The Traill-Steele District members were kind enough to attend a joint meeting with the Grand Forks District, at which I was a guest. The Sheyenne Valley District joined the Cass County District when I met with the latter society. The meetings were well attended. Discussions were largely of legislative problems, in which a very noticeable increase in interest and, I hope, participation was initiated. Some societies are now setting aside one or two meetings a year to be devoted entirely to matters of concern to medicine and the society. I believe this a very wise idea.

In concluding this report, I have two or three suggestions for your consideration.

1. I believe consideration should be given toward approving and allotting funds to provide the entire membership of the society with the *Handbook* prior to the annual meeting. Members are being asked to participate more fully as individuals in medical affairs and, possibly, to write a larger check for the dues. It is only right that they should be better informed as to the proceedings of the society.

2. Although the proceedings of the annual meeting can be read by all in *THE JOURNAL-LANCET*, I believe that it would be a distinct advantage (we have tried it this past year in the Devils Lake District) to have a district society meeting shortly after the annual state meeting, so that the delegates and councillors may report while important matters are still fresh in their minds.

3. If we are to retain *THE JOURNAL-LANCET* as our official magazine, and I am convinced it is doing a good job for us, then I feel that we should insist on having larger recognition on its front cover, stating that it is our official publication. I would think that we might well ask for, say, half of the front cover blocked off in a different color and entitled with whatever descriptive name we might approve, such as *North Dakota Medicine* or *Medical Journal of North Dakota*.

4. For several years, I have been very conscious of the vacuum that a man steps into when he is elected second vice-president of the society and at that time drops out of active participation in much of the society's work. This is not right because the services of these men are largely lost for three years, and, at the same time, they are apt to lose track of what is going on in the society. I believe that they should officially be given certain duties within the structure of the organization, so that they may keep abreast of the times, or that the tradition of their dropping out of other offices be changed, at least until they become president-elect.

I have perhaps reported herewith at too great length, but it has truly been a big year. For me, it has been a great privilege and honor to have been able to act as your president and official representative. I can only hope that I have been able to serve the association in a satisfactory manner and that my efforts may have added in a small way to the conduct of its various interests. I wish to thank Mr. Lyle Limond, our executive secretary, for his able and willing cooperation over and above the call of duty and to thank the many members of this society for their willing acceptance of responsibilities in com-

mittee appointments and so forth. Last, I wish to extend best wishes to your new president for the coming year and ask that you fully cooperate with him and assist him at all times.

J. C. FAWCETT, M.D., President

REPORT OF THE SECRETARY

MEMBERSHIP: The total membership for 1959 was 448. Of this number, 418 paid the regular membership fee, 4 were on a retired or limited basis, 24 were honorary members, and 2 were carried on a complimentary basis due to military service and age. Eight members passed away during the year, and several have left the state. New members, however, are being steadily added to our roster.

Table 1 shows the annual membership for the past five years.

TABLE 1
COMPARISON OF ANNUAL MEMBERSHIP

	1955	1956	1957	1958	1959
Paid memberships	387	380	395	403	418
Honorary memberships	14	16	18	16	24
Retired and limited	12	12	9	9	4
Dues cancelled, military service and age exemption	3	8	6	5	2
Total	416	416	428	433	448

Table 2 shows the annual dues for 1960, which have been coming in very slowly. There are still many members who have not as yet paid their 1960 dues, and the district medical society secretaries and councillors are urged to use every possible means to collect the dues of these delinquent members.

TABLE 2

	April 19 1956	May 1 1957	April 15 1958	April 15 1959	April 13 1960
Paid-up members	334	328	313	309	335
Honorary members	16	18	16	20	22
To be honorary	6	2	3	4	5
Dues cancelled, military service	5	5	3	3	2
Limited	1			1	2
Retired	6	7	3	1	1
Total	368	360	338	338	367

STATE ASSOCIATION MEMBERSHIPS

1959:	Regular	Retired	Limited	Comp.	Honorary
First	88			1	1
Second	28				2
Third	72			1	4
Fourth	63		1		5
Fifth	7				1
Sixth	70		1		6
Seventh	34	1	1		2
Eighth	19				
Ninth	26				3
Tenth	11				
Total	418	1	3	2	24

1960:	Regular	Retired	Limited	Comp.	Honorary
First	69			1	1
Second	21				2
Third	55			1	4
Fourth	40				3
Fifth	7				1
Sixth	68	1	2		6
Seventh	30				1
Eighth	17				
Ninth	19				4
Tenth	9				
Total	335	1	2	2	22

AMA GENERAL MEMBERSHIPS

	1959	1960
First	88	70
Second	30	23
Third	75	59
Fourth	70	43
Fifth	8	8
Sixth	76	75
Seventh	37	30
Eighth	19	17
Ninth	27	23
Tenth	12	9
Total	442	357

Of course, the association wishes that all doctors practicing in North Dakota would become members of our group. This, of course, has not happened in the past and probably will never happen entirely, but, nevertheless, it behooves each of us to attempt to have our fellow physicians join and take an active part in the medical association.

As was noted previously, the dues this year have been coming in rather slowly. Failure to pay these dues when they become due on January 1 creates unnecessary work for the secretaries of the constituent societies, and the resultant uncertainty is disconcerting to the state office. It is suggested that each member add to his other New Year's resolutions the resolve to pay medical society dues during the first week of each new year.

F. D. NAEGELI, M.D., Secretary

REPORT OF THE EXECUTIVE SECRETARY

GENERAL COMMENTS: Your executive secretary attended several state, regional, and national meetings in behalf of the association.

The routine of the headquarters office continues to show an increased tempo as each year passes. This increase is due to increased emphasis in the areas of medical economics, legislation, medicare, public relations, and general services to the members.

The affairs of your state office remain in the capable hands of Mrs. G. K. Fremming, office secretary. Part-time help is employed when the occasion warrants it.

LEGISLATION: Our greatest efforts in this area were on the national scene in combating the Forand bill. The members of this association and the woman's auxiliary responded in a most creditable manner in opposing the Forand bill through the use of telegrams, letters, and resolutions.

This coming summer and fall should be spent in readying ourselves for the political campaigns, the elections, and the opening of our State Legislative Assembly in January 1961.

PHYSICIANS' PLACEMENT SERVICE: Twenty-six North Dakota communities and 10 physicians or groups are on file in the office in regard to requests for a physician or additional physicians.

The 26 communities are as follows: Anamoose, Ashley, Belfield, Buffalo, Edmore, Enderlin, Esmond, Flasher, Fordville, Forman, Grenora, Hatton, Killdeer, McHenry, McVie, Mandan, Medina, Milnor, New England, Page, Pembina, Rutland, Sharon, Strasburg, Willow City, and Wyndmere.

U.N.D. MEDICAL SCHOOL SCHOLARSHIPS: The 1959 winners of the association's scholarship prizes, totaling \$500, offered at the School of Medicine were as follows: anatomy, first year, Gyman C. Okeson; physiology and pharmacology, Charles Halgrimson; Microbiology, Charles Halgrimson; pathology, second year, Roy R. Schale; and highest scholastic average, first year, John W. Gilsdorf.

FINANCE: The treasurer's report continues to show an improved balance. The goal of having one year's operating budget in reserve is being maintained as it should be in the interests of good business practice.

Receipt of dues continued to be slow as in years past, as will be noted in the following listing of April 15, 1960.

<i>District society</i>	<i>Number of unpaid members</i>
First	9
Second (Devils Lake)	4
Third (Grand Forks)	20
Fourth (Northwest)	25
Fifth (Sheyenne Valley)	0
Sixth	3
Seventh (Stutsman)	3
Eighth (Kotana)	0
Ninth (Southwestern)	6
Tenth (Traill-Steele)	1
Total	71

MEDICARE: The Dependents' Medical Care Program (Medicare) commenced on December 7, 1956. Up to January 1, 1960, 2,573 claims had been processed by this office. The total sum paid to North Dakota physicians as of December 31, 1959, amounted to \$173,246.79. Each claim for services rendered averages \$67.19.

On April 1, 1960, we signed a new Medicare contract with the federal government. This contract restores many of the coverages which were deleted from the program on October 1, 1958. With a few exceptions, we are now back to the Medicare program as instituted on December 7, 1956.

THOUGHTS FOR THE FUTURE:

1. Serious thought must now be given to plans for the association's diamond anniversary scheduled for Bismarck in 1962.

2. Interest should remain high in the field of legislation affecting organized medicine.

3. Greater interest should be given and more definitive action should be taken by the association in the problems of our aging population.

4. To create greater committee interest, thought should be given to the suggestion that the president-elect and

the two vice-presidents accept the responsibility for certain committees.

5. Thought should be given now to an expanded program in the area of public information and education (public relations) in cooperation with North Dakota Blue Shield. It is felt that Blue Shield could legally and legitimately expend some of its funds, along with funds from this association, which are needed to underwrite a cooperative program from the standpoint of salaries, travel, printed material, and so forth.

Such a stepped-up program of public information and education could include the following: (a) newsletter to all daily and weekly newspapers, (b) speakers bureau, (c) radio and TV programs, (d) exhibits at state and county fairs, (e) press, radio, and TV conferences, (f) public forums, (g) Careers Day (night) program support, (h) health columns in North Dakota newspapers and magazines, (i) paid advertising on occasions, and (j) film library.

Leadership and coordination for such a program would be supplied by this association.

6. Serious consideration should be given to having some of the association members visit the legislators for a day or two during the 1961 legislative session, even though we would not be supporting or opposing any bills at the time of the visitations.

ACKNOWLEDGEMENTS: I wish to express my sincere appreciation to our president, John C. Fawcett, M.D., for his efforts in behalf of this association. Dr. Fawcett was ever willing to leave his busy practice to attend district society meetings and other meetings of importance to this association.

My sincere thanks also go to those other members with whom I have had occasion to work during this past year in the association's several programs.

LYLE A. LIMOND, Executive Secretary

REPORT OF THE CHAIRMAN OF THE COUNCIL

The Council of the North Dakota State Medical Association held its regular spring meeting May 2 and 3 at the Prince Hotel, Bismarck. The interim meeting was held December 12 and 13 at the Gardner Hotel, Fargo.

All of the district councillors and the councillor-at-large were present at the council meeting held May 2. Others present were our president, Dr. O. A. Sedlak, and Drs. E. H. Boerth, R. H. Waldschmidt, E. J. Larson, L. W. Larson, G. A. Dodds, J. C. Fawcett, C. M. Lund, R. D. Nierling, and Mr. Lyle A. Limond.

Minutes of the previous sessions were approved.

Dr. L. W. Larson discussed the report of the American Medical Association Commission on Medical Care Plans and the questionnaire which was sent out to all state medical associations. A motion was passed that the council request the House of Delegates give favorable consideration to a resolution stating that the North Dakota State Medical Association is in sympathy with this report as a whole.

Dr. L. W. Larson expressed his gratitude for the support that has been given him by the North Dakota State Medical Association on his anticipated candidacy for the presidency of the American Medical Association.

Dr. E. J. Larson, treasurer, informed the council that he had invested \$5,000 of the association's funds in certificates at a local bank. However, he stated that government bonds would earn more interest and that he would suggest that this amount be placed in government bonds at the time the certificates mature. The council passed the motion that Dr. E. J. Larson be instructed to invest

the \$5,000 in government bonds when the certificates mature.

There was a discussion regarding delinquent membership dues, and ways and means of collecting these dues were taken up.

Dr. C. H. Peters discussed the negotiations carried on by the Committee on Medical Economics. He discussed at some length the difficulties the committee is having in coming to an agreement with the State Welfare Board on the Relative Value Schedule. The board brought up 4 counterproposals which are, to wit:

1. It wants an advisory committee. The question is, how should that committee be set up? Should an advisory committee of the state medical association be appointed, or should it be set up by the welfare board, with members selected from the association? The board suggested that the state medical association give it the names of 6 physicians and it would pick 3 of them to be on its committee.

2. Another problem is the psychiatric care cases. The welfare board asked that this association provide names of doctors in North Dakota who are trained to provide psychiatric care. We refused to do this.

3. The board wants some type of operating procedure to gather information from local district societies regarding welfare problems, and it would like to have this channeled into a committee that would be set up. I think there is value in this. If we are going to have a committee to the public welfare board, it should be a state medical association committee composed of at least 1 man from each district in the state.

4. This item brought up the rates. The board proposed no increase per se in schedule, but a 15 per cent increase was suggested across the board in the schedule, pending the approval of the welfare board. This suggestion came from the executive director. The difficulty with the 15 per cent increase is that there are so many inequities in the old fee schedule.

We want to be able to continue with further negotiations, and, in our recommendations to the House of Delegates, we will suggest that the negotiations be continued to some conclusion to at least iron out, if possible, some inequities that exist in the present fee schedule. In view of the difficulties, we felt that the House of Delegates should be fully informed, and I took the liberty of asking Mr. Carlyle Omsrud to appear before the House of Delegates this afternoon to discuss this problem and be prepared to answer questions. I hope the House of Delegates will leave the gate open so that we can continue negotiations. I believe that the president should appoint a brand new committee, as it may work out better for everyone concerned.

Mr. Limond discussed a letter which he had received from the American Association of Medical Assistants asking that the North Dakota State Medical Association approve of a chapter in North Dakota on medical assistants. After much discussion, the council recommended that this request be given favorable consideration by the Committee on Resolutions.

Mr. Limond asked for a ruling regarding the seating of delegates from district societies who have not paid their dues. Dr. Dodds informed him that the House of Delegates would not refuse to seat a delegate in these circumstances.

Mr. Limond also asked for a ruling as to whether the association should carry as members doctors who leave the state for a year's leave of absence to take postgraduate work. The council ruled that this should be done,

since the man is not practicing medicine in any other state and is improving himself to aid his community when he returns.

After discussing the contents of a letter received regarding a Toxicological Laboratory in the state, motion was made and passed by the council that it go on record as approving the eventual establishment of the Toxicological Laboratory at the University of North Dakota and that this matter be referred to and acted upon by the Committee on Resolutions.

A resolution was brought up which was received from the medical society of Wisconsin regarding the development of an annual postage stamp on safe transportation. A motion was passed by the council to approve of this resolution and pass it on to the Committee on Resolutions.

A resolution was discussed regarding the appointment of a medical doctor as the state health officer. A motion was passed that this resolution be referred to the Committee on Resolutions, with a recommendation that it act favorably upon this resolution.

A resolution was received by the council regarding the appointment of a medical doctor as superintendent of the Jamestown Hospital. After much discussion, a motion was passed that the resolution be referred to and acted upon by the Committee on Resolutions.

The second session of the council was held on May 3, 1959, and, in addition to the councillors, was attended by Drs. E. H. Boerth, J. C. Fawcett, C. M. Lund, O. A. Sedlak, R. H. Waldschmidt, R. D. Nierling, and Mr. Lyle A. Limond.

The National Chamber of Commerce requested an increase in council dues, amounting to a total of \$150. A motion was passed that this payment be made.

Drs. Foster and Hamz, members of the Committee on Scientific Program, appeared before the council and stated that they were having problems in procuring speakers because they were not able to offer the speakers an honorarium. After discussion, a motion was passed by the council to allow the Committee on Scientific Program \$500.00 for honoraria for obtaining out-of-state speakers and for miscellaneous expense in procuring the speakers.

A motion was passed that the annual reports of the councillors of the district cover from March to March.

Dr. Peters discussed the news release on the resolutions passed in the afternoon by the House of Delegates concerning the State Hospital and an advisory committee to the Board of Administration. After a lengthy discussion, Dr. Peters moved and Dr. Toomey seconded that a committee from the council be appointed to formulate a press release to explain the action of the House of Delegates and that this news release be given to the council, after a fifteen minute recess, for its approval. Motion passed. Dr. Gilsdorf appointed Drs. Rodgers, Vandergon, Peters, and himself to the committee to formulate the press release.

When the Council reconvened after fifteen minutes, the press release met with the approval of the councillors. Dr. Craven moved and Dr. Rodgers seconded that the news release be accepted. Dr. Toomey then moved that this advisory committee, which is to meet with the governor on Monday morning, be empowered to release and delete any part of this news release at its discretion. Dr. Borland seconded this motion. Motion was passed.

The following is the final press release given to the press Monday afternoon after the morning meeting with the governor.

News Release

The House of Delegates of the North Dakota State Medical Association, Sunday, May 3, 1959, passed the following Resolutions:

RESOLUTION

Whereas, the primary interest of the North Dakota State Medical Association is in the best possible medical care for the citizens of North Dakota,

Now, therefore, be it resolved that the North Dakota State Medical Association recommends that the superintendent of the state institutions for patient care at Dunseith, Grafton, and Jamestown be a qualified doctor of medicine.

RESOLUTION

Whereas, the North Dakota State Board of Administration is charged with the management of several of our state institutions, the operation of which involves problems of a medical nature, and

Whereas, the North Dakota State Board of Administration does not have, at present, an advisory committee on medical problems,

Now, therefore, be it resolved that the president of the North Dakota State Medical Association shall, upon request by the North Dakota State Board of Administration, appoint a liaison committee to assist and advise that board in matters of a medical nature which may come under its jurisdiction.

In light of the above resolutions, the president of the North Dakota State Medical Association has been authorized by the association's House of Delegates to appoint a liaison committee to assist and advise the Board of Administration and/or the governor in matters of a medical nature, should they so desire.

It is the earnest hope of the North Dakota State Medical Association that by a cooperative effort, the best interest for the care and treatment of the mentally ill of North Dakota will be served.

The members of the delegation of physicians who visited the governor Monday morning, May 4, concerning the Jamestown situation and the official actions of the North Dakota State Medical Association's House of Delegates, are as follows: John C. Fawcett, president, Devils Lake; O. A. Sedlak, immediate past president, Fargo; C. M. Lund, president-elect, Williston; G. A. Dodds, speaker of the house of delegates, Fargo; R. D. Nierling, treasurer, Jamestown; C. H. Peters, secretary of the council, Bismarck; and V. G. Borland, member of the council, Fargo.

The chairman of the council called for the election of officers for the following year. The following nominations were made and approved: chairman, Dr. A. R. Gilsdorf; vice-chairman, Dr. V. G. Borland; secretary, Dr. C. H. Peters.

The chairman of the council appointed the executive committee, which consists of the 3 officers of the council: Dr. A. R. Gilsdorf, Dr. V. G. Borland, and Dr. C. H. Peters.

At the interim council meeting on December 12, 1959, all councillors were present with the exception of the councillor from the eighth district. Others present were: Drs. J. C. Fawcett, C. J. Glaspel, Walter Pretorius, E. H. Boerth, G. A. Dodds, R. M. Fawcett, C. M. Lund, and Mrs. Margaret Fremming and Mr. Lyle A. Limond.

Reading of the minutes of the previous meetings was dispensed with.

Dr. Walter Pretorius of the North Dakota Public Health Department was introduced and presented a secondary rheumatic fever program. This program offers 3 primary services: (1) low cost medication, (2) regular checkups by Public Health Nurses, and (3) periodic reminders to the patients to return for care.

Dr. R. M. Fawcett advised the council that this program had been discussed by the Cardiovascular Committee, and that it felt that this was a favorable program and recommended that the North Dakota State Medical Association give its approval, pending approval by the

Pharmaceutical Association and the Heart Association. After a lengthy discussion between the councillors, Dr. Vandergon made the motion that the program be tabled for the present time. Motion was seconded by Dr. Toomey but lost. Dr. Pretorius was excused. It was felt that the program had many worthwhile points and that more time should be given to the study of its advantages. A motion was made that the matter be referred back to the Cardiovascular Committee for action at the May meeting.

Dr. C. H. Peters next reported on the work of the Medical Economics Committee concerning negotiations with the Public Welfare Board. The board had offered a 15 per cent increase on the old schedule, which appeared to be satisfactory if the 15 per cent was applied to the schedule after certain inequities had been adjusted to the satisfaction of the association. The negotiating team met Mr. Ralph Atkins on December 12 in Fargo to adjust these inequities. A committee will review the schedule on a regular calendar basis and will meet monthly, if possible, and never less than quarterly and will meet the Public Welfare Board once a year. It was felt that this mediation committee was needed, as both sides have complaints and problems that they will be able to resolve. If a deadlock should occur, 1 member from the Mediation Committee and 1 from the Welfare Board will select one other person to solve the problem. It was felt that this would be acceptable to the Welfare Board. A motion was passed that the medical economics report and the agreement with the Welfare Board be approved. Dr. Peters reported that both the Indian Bureau and Blue Shield had accepted the relative value fee schedule. The Workmen's Compensation Bureau was still studying the schedule, and the association is, at this time, just asking for a survey. There had been only 1 meeting with Vocational Rehabilitation, and it was felt that it would continue to pay the usual, private fee of the physician.

The Council congratulated Dr. Peters and the various members of his committee on their excellent work.

Dr. C. J. Glaspel, secretary of the North Dakota State Board of Medical Examiners next briefly reported on the activities of the board. It was deemed desirable that the House of Delegates of the association should be given a report on the various activities on a state level, together with résumés of national meetings. His discussion centered around the many problems presented by the foreign graduates and the accomplishments of the educational council in this regard.

The next item on the agenda was the discussion of Blue Shield and Blue Cross problems. Dr. Sedlak informed the Council that Blue Shield problems were few. The most serious problem confronting the Blue Cross program is the abuse of the prepayment plan, and greater cooperation between the physicians is needed to help eliminate one- and two-day stays in hospitals. It was felt that the doctors' public relations are not good, and it is most desirable that there be a much greater understanding between the physician and the public. It was felt that all doctors should be more interested in politics on all levels. The Council was asked to consider the possibility of some one person or agency to handle public relations for both the association and Blue Shield. Legislative problems in 1961 would undoubtedly prove less troublesome if the state and local legislative committees could create increasing interest in most phases of politics. A motion was passed that the Council approve the development of a Public Relations Committee coordinated with the public relations program of Blue Shield and to develop a public relations department.

Mr. Limond presented several methods which might be used in molding public opinion. These included radio, TV, press, and monthly news releases. It was thought desirable that billings sent out from physicians' offices could include some type of a leaflet. This method would bring information to thousands of North Dakota residents. Foreseeing a long-term program, Dr. Borland proposed that the dues of the members of the North Dakota State Medical Association be raised so that funds would be available for such a public relations campaign. Dr. Borland moved that up to \$15,000 out of the present surplus in the association fund be used in 1960 for a public relations program. Dr. G. Christianson seconded the motion. Much discussion followed concerning this motion, and it was Dr. Peters' consensus that we use the present budget earmarked for public relations, study the plan more carefully, and hold off action until the next meeting of the council in May 1960. Dr. Borland withdrew his motion.

It was felt desirable that a new committee composed of 3 members of the Council, namely, Drs. Vandergon, Borland, and Pederson, be formed to make plans on a district level.

Motion was passed that this committee, already agreed upon, move immediately to make a survey of action for plans to bring to the House of Delegates in May. A motion was then passed that a similar request be made for Blue Shield to be brought to the Resolution Committee in May.

There was discussion regarding closer association between the North Dakota State Medical Association and the Blue Cross Board. It had been suggested to the council that the association recommend the names of 5 physicians to the Blue Cross Board, asking the board to select 3 of them to form a liaison committee. A motion was made and passed that no action be taken on this suggestion at this time.

Mr. Limond gave a brief résumé of the Medicare program as it is today in North Dakota. He advised the council that a directive had been received from the Department of the Army that the old, more extensive program would be in force again in 1960. The history of Medicare in North Dakota has been exemplary.

The association's Group Life Insurance Program was next discussed. Oscar Hanson, the state agent, had reported that 150 members of the association were now participating in the program and that it was moving well and without complaints. The council was advised that the Union Central Life Insurance Company was holding a rebate check in the sum of \$6,958.29 on deposit at 3 per cent per annum and that another check would be due the policy holders in April. Mr. Limond asked the council what it thought should be done with the money, whether it should be refunded to the individual policy holder or possibly used to increase the amount of their insurance. A motion was passed that a survey be made to the individual policy holders as to their wishes in the matter.

A motion was passed that the liability policy holders also be surveyed in regard to their satisfaction with that plan and that current information also be obtained from Mrs. E. A. Shepler, agent.

The budget for 1960 was discussed and approved by the council.

The council took the following actions concerning the North Dakota Physicians Service:

1. Concurred that the attending physician (private practitioner) is responsible for the outpatient in a hospital in which there are interns and/or residents.

2. Concurred in the recommendation of the Medical Economics Committee of the North Dakota State Medical Association that Blue Cross and Blue Shield be advised that policies of greater than \$25 deductible and with a larger coinsurance feature should be introduced to the public.

3. Concurred in the recommendation of the Medical Economics Committee to Blue Cross and Blue Shield that the Grievance Committee of the North Dakota State Medical Association is available to these organizations to review complaints that they may have against members of this association. It was further suggested that, when fully documented evidence of unethical conduct or abuse in overutilization of prepaid medical plans has been found, these 2 organizations make use of the association's Grievance Committee.

The Council also took the following actions concerning the request of the North Dakota Hospital Service Association.

Tabled for the time being the proposition that the North Dakota State Medical Association request of Blue Cross the privilege of recommending the names of the 3 physician members to the Blue Cross Board of Trustees.

A motion was passed that expenses of the alternate delegate of the American Medical Association be paid for both sessions. It was noted that a budget item is now allocated for conferences and delegates, and this budget would still be adequate to allow this additional expense.

Dr. Pederson, chairman of the State Association's Committee on Aging and Rehabilitation, made a brief report to the Council. Money will be available to North Dakota for a survey on the needs of the aging in the state, with the compiled information taken to the White House Conference in January 1961. It was suggested that a letter be directed to John Davis, governor of North Dakota, containing the names of the officers, councillors, and members of the state association committee, together with the names of W. A. Wright, M.D., and L. W. Larson, M.D., with the request that the governor appoint physicians from this listing as representatives to the state conference.

A motion was passed that our president, Dr. J. C. Fawcett, write such a letter to the governor.

It was suggested by Mr. Limond, and agreed to, that the House of Delegates give thought to a resolution stating that physicians in North Dakota believe in taking care of all persons regardless of ability to pay. This philosophy is time-honored and part of the ethics of medicine.

Dr. Halliday spoke briefly concerning *Medical Milestones*. He felt that it would be most desirable for the association to appoint a historian for the society and that the members of the Necrology Committee should direct the program of such a historian. It is hoped to have all corrections in the *Milestones* completed by the time of the May meeting, so that the committee can review them.

Additional duties of the council. It is to be noted on page 32, chapter XIII, section 1 of the Constitution and Bylaws of the North Dakota State Medical Association, dated April 1958, "Collectively, the council shall be the Mediation Committee of the association. The chairman of the council shall act as chairman of the Mediation Committee. In some instances, the action of the council as a Mediation Committee will closely parallel its activities as a board of censors of the association. A majority of the members of the Mediation Committee shall constitute a quorum for the transaction of business."

The Mediation Committee for the state association serves also as the Grievance Committee. This committee

has received communication from Mr. Al Doerr, secretary of the North Dakota Pharmaceutical Association, 1208 South Highland Acres Road, Bismarck, requesting a "hearing with the Grievance Committee regarding the concern of the pharmacy profession as to the growing number of clinics within North Dakota having associated pharmacy self-owned or of joint venture." Mr. Doerr concluded with, "May I invite you to a joint meeting in which we make a sincere effort to resolve this problem. I would like to believe that we should be able to make substantial progress hereon and, in any event, initiate a program. You may be sure we appreciate your cooperation."

Members of the Council, acting as the Grievance Committee, have corresponded with Mr. Doerr and he and his group have been invited to attend the council meeting at the time of our spring session in Grand Forks for discussion on the grievance.

Adjournment of this council meeting at 12:50 A.M. on Sunday, December 13, 1959, completed our formal meetings for the year.

A. R. GILSDORF, M.D., Chairman of the Council

REPORTS OF COUNCILLORS

First District

The First District Medical Society held 8 meetings during the fiscal year from March 1958 through February 1959. No meetings are held during the months of May, June, July, and August. The meetings are held in the Town Hall of the Gardner Hotel, Fargo, on the fourth Tuesday of each month.

On March 24, 1959, the society decided to devote this meeting to several business matters. Henceforth, the meeting in March each year will be devoted to discussion of business matters in general and to instructions for our delegates to the state meeting. In the early fall, a similar meeting will be set aside for the delegates to report back to the society on the action taken at the state medical meeting. Dr. Frederick Hofmeister, of the Department of Obstetrics and Gynecology at Milwaukee Hospital, read a paper on "The Papanicolaou Smear in Private Practice."

The meeting on April 27 was devoted to the annual Darrow-Long lectureship, sponsored by the Dakota Clinic, and was held at the Top of the Mart in the Frederick-Martin Hotel in Moorhead. The speaker for this occasion was Dr. George Herrmann, professor of medicine at the University of Texas, who gave a lecture on "Modern Concepts of Atherosclerosis and its Management."

On Tuesday, September 29, an excellent panel program was given by 4 representatives of the Cass County Bar Association. This included presentation of many of the interrelated problems between physicians and lawyers and was very much appreciated by the society. A lively discussion followed.

Scientific matters were eliminated from the program of the October 21 meeting. The delegates to the State Medical Society meeting in May made a report to the Society. Much of the discussion centered around the problems of welfare patients. It was decided that a local committee on public relations be reactivated and that the first district medical society allow this committee an annual budget of \$500.

At the meeting held on November 24, a very interesting scientific program was presented by Drs. Dodge and Thomas of St. Louis, who reported on problems connected with the use of the artificial kidney. Drs. Pierson and Morton of Bismarck were visitors at this meeting.

At the meeting on December 15, committee reports were made by Drs. Lancaster, DeCesare, Armstrong, and Borland. Officers elected for 1960 were: Dr. Robert Rogers, president; Dr. Howard Hall, vice-president; and Dr. M. H. Poindexter, secretary. Dr. Ralph Weible was appointed to Board of Censors.

The delegates to the state meeting were elected as follows: Drs. Pray, DeCesare, Burt, Beithon, Melton, and Macaulay. The alternates selected were: Drs. Norum, Jaehning, Murray, Schneider, Christoferson, and Wold. The members stood for a moment of silence out of respect to Dr. Donald Olson who died as a result of an automobile accident.

At the regular meeting on January 19, Dr. Gordon Magill was unanimously elected to membership. Several committee reports were made and the speaker of the evening, Dr. Philip Osmundson of the Department of Peripheral Vascular Diseases of the Mayo Clinic, spoke to us on "The Management of Arteriosclerosis Obliterans."

At the meeting on February 23, committees were appointed and announced by Dr. Rogers. Drs. C. W. Nelsenmoe and John Sessums were elected to membership. The motion was made and seconded that our delegates to the state meeting voice their disapproval of the care of nonservice-connected disabilities in the veterans hospitals. The delegates were also instructed to favor the initiation of a prenatal mortality study in the state. The guest speakers were Dr. John Fawcett, president of the state society, and Dr. Clifford Peters of Bismarck. Several guests were present from Valley City at this meeting.

Membership of the district is as follows: active members, 87; retired members, 2; honorary members, 0; limited members, 6; service members, 0; new members added during the year, 3; members transferred, Dr. Eleanor Crim, Fargo, to India; and Dr. Erwin Johnsrude, Fairmount, contemplated residence unknown; deceased members, Dr. Donald Olson; and nonmembers residing and practicing in the First District, Dr. H. B. Waydeman, Hunter, and Dr. Jerome P. Haeger, Hankinson.

V. G. BORLAND, M.D., Councillor

Second District

The Devils Lake District Medical Society held 8 regular meetings during the past year from February 1959 through January 1960. All of the meetings were well attended. All of the practicing physicians in the area are members of the society.

Officers for the fiscal year of 1960 are as follows: president, Dr. R. Donald McBane, Devils Lake; vice-president, Dr. Stuart Cook, Rolette; and secretary-treasurer, Dr. L. F. Pine, Devils Lake. Alternate delegates for the past year were elected delegates for the present year: Drs. D. W. Palmer, Cando; and J. H. Mahoney, Devils Lake. Alternate delegates: Drs. C. H. Hiltz, Cando, and G. W. Seibel, New Rockford. Censor: Dr. J. Terlecki, Minnecaukan.

Out-of-town speakers presented the scientific programs at most of the meetings, including the following:

February 5. Dr. Cuadrado, superintendent of the North Dakota Tuberculosis Sanatorium, gave a very interesting talk on the aspect of endeavoring to uncover the rampant type of tuberculosis, stressing particularly the usage of the Mantoux test with PPD.

March 5. Dr. Poindexter of Fargo presented a paper on "Staphylococcal Infections in Hospitals."

April 2. Dr. Joseph N. Kiely of the Mayo Clinic presented a paper entitled "Chemotherapy of Malignant Diseases."

May 21. There was no out-of-town speaker at this meeting nor was a strictly scientific paper presented. Instead issues brought up at the state meeting in Bismarck were considered. Dr. G. W. Toomey, councillor from the Second District, gave a report on the Council activities. Dr. W. R. Fox, delegate, presented his report on the House of Delegates and legislation talked over at the state meeting. Dr. John Fawcett, the new state president, gave his first address of the year to this society, of which he is a member. Dr. Mahoney reported from the Economics Committee.

September 10. No out-of-town speaker was obtained for the scientific program, and the meeting was devoted entirely to current, nonscientific problems.

October 1. The program was presented by Mr. Wm. P. Lanier, Jr., Fargo, whose topic was "Medical-Legal Relations Between Physicians and Attorneys." The dentists were invited to this meeting, and some very pertinent subjects were discussed by Mr. Lanier. The scientific program was postponed to December because of inclement weather.

December 3. The speaker for the scientific meeting was deterred by inclement weather again. However, Dr. O. W. Johnson was present at the meeting and spoke against bureaucratic socialism via the Forand bill. President, Dr. John C. Fawcett, made some very pertinent remarks in regard to the medical profession being the last bulwark of economic independence.

January 14. Dr. L. G. Pray of Fargo spoke on "Psychiatric Problems in Children."

Fiscal problems were discussed at great length during the year, including Blue Cross-Blue Shield, the Forand bill, sponsoring of the Women's Auxiliary essay contest, and so on. Considerable time was spent at meetings discussing the importance of physicians in each locality contacting and cultivating their legislators, thereby providing us with better public relations and better contacts when unfavorable legislation appears.

It is felt that membership, attendance, and harmony in the Second District have been at least average, if not above.

G. W. TOOMEY, M.D., Councillor

Third District

This society lost 4 members in 1959 and added 4 during the year. We have a total membership of 77, of whom 5 are honorary members. Five doctors in the district are eligible for membership and are not members. Some of these belong to other districts, and some are teaching in the medical school.

The society's financial condition is sound. There have been no ethical problems brought to my attention during the past year.

Eight meetings were held during the year 1958-1959, with attendance running from a low of 29 to a high of 45.

Good scientific programs were held at each meeting with both local and out-of-district speakers presenting programs.

In addition to the scientific programs, the president of the association, Dr. John C. Fawcett, Dr. O. W. Johnson, and Mr. Lyle Limond discussed the Forand bill at one meeting. At another, Dr. O. A. Sedlak, immediate past-president of the association, discussed "Current Legislative Problems in North Dakota."

A legislative committee has been appointed and a diabetic drive has been approved and sponsored.

Officers for 1960 are: president, Dr. Charles B. Porter; vice-president, Dr. N. J. Kaluzniak; and secretary-treasurer, Dr. William P. Keig.

P. H. WOUTAT, M.D., Councillor

Fourth District

January 22, 1959. The first meeting of the year was held at the Clarence Parker Hotel. After the election of officers, Dr. Reiser of the University of Minnesota spoke on the "Conservative Treatment of Anuria."

February 26, 1959. This meeting was also held at the Clarence Parker Hotel. Forty-two members were present. Dr. John Moe of the University of Minnesota spoke on "Deformity of the Feet in the Newborn."

March 26, 1959. Meeting was held at the Minot Country Club, with 30 members attending. Miss Alexine Grenz was the speaker. Her topic was "The Activity of the Vocational Rehabilitation Program."

April 23, 1959. Meeting was held at the Minot Country Club, and 36 members attended. Dr. Dehlin from the Mayo Clinic gave a talk on "Exfoliative Cytology."

June 25, 1959. Meeting was held at St. Joseph's Hospital. Dr. Malcolm McCannel of Minneapolis, who had recently returned from observing and doing work on the eye in India, gave a most interesting talk on "Modern Medicine in the Far East."

September 24, 1959. Meeting was held at the Clarence Parker Hotel. Dr. Heidorn of Minot was the speaker. His topic was "Electrocardiography in Asymptomatic Adult Males."

October 22, 1959. Meeting was held at the Minot Country Club, and 45 members were in attendance. Dr. M. G. Peterman of Milwaukee gave a most informative talk on "Factors of Brain Injury in the Child."

November 19, 1959. Meeting was held at the Minot Country Club. Principal speakers of the evening were Drs. O. W. Johnson of Rugby and Carroll Lund of Williston, who discussed at some length the Forand bill.

January 28, 1960. Meeting was held at Trinity Hospital, and 42 members were present. Dr. Pratt of the Mayo Clinic gave a most interesting talk on "Abdominal Pain."

February 25, 1960. Meeting was held at Trinity Hospital. Dr. Tom Chin of the Public Health Department of Atlanta, Georgia, spoke on "Virus Research Problems." The election of officers, which had been postponed from the January meeting, was held. Dr. Sam Shea was re-elected president, and Dr. Lloyd Giltner was re-elected secretary-treasurer. Dr. Darwin L. Kohl was elected vice-president, replacing Dr. Wm. Kitto, who has moved to Ann Arbor, Michigan.

During the year, 8 new members were admitted to the society: Drs. David Halliday, R. H. Whittelsey, O. M. Simms, E. B. McConville, Jens Sahl, E. Bernudez, John Anthony, and Brian Briggs.

One member, Dr. C. F. Schnee, transferred from the society. Two members, Drs. J. S. McArdle and A. D. McCannel, died. Total membership is 76; active members number 71; and 5 are retired or honorary.

D. H. HALLIDAY, M.D., Councillor

Fifth District

The Sheyeme Valley Medical Society held 5 dinner meetings during the past year.

The first meeting was held at Mercy Hospital on May 21, with Dr. G. B. Magill of Fargo as guest speaker. Dr. Magill discussed "Chemotherapy of Carcinoma."

On June 17, a meeting was held at Mercy Hospital, and the Southwest Blood Bank program was presented by Mr. William Burt of Fargo. After discussion, the program was approved.

The next meeting was again held at Mercy Hospital on October 21. The program consisted of a showing of a film on "Clinical Problems in Diseases of the Chest."

On January 15, 1960, a meeting was held at Sheyenne Memorial Hospital, at which time the following officers were elected: president, Dr. N. A. Macdonald; vice-president, Dr. J. W. Goven; and secretary-treasurer, Dr. C. J. Klein. Delegate: Dr. N. A. Macdonald. Alternate delegate: Dr. C. J. Klein.

On March 19, our meeting was again held at Sheyenne Memorial Hospital. Discussion was devoted to "The Rehabilitation of the Stroke Patient."

Members took advantage of the hospitality of nearby societies to attend scientific meetings.

Total active membership in the society is now 7, with 1 retired member. Dr. K. M. Wakefield of Cooperstown transferred to the Traill-Steele Medical Society.

G. CHRISTIANSON, M.D., Councillor

Sixth District

The Sixth District Medical Society held 6 meetings during the calendar year of 1959. Additional meetings were held in February and March 1960 to bring the councillor's report up to date prior to our May meeting.

The first meeting was held on January 6, 1959. The president, Dr. O. A. Sedlak of Fargo, was introduced and discussed briefly some of the problems confronting the medical profession in North Dakota. Mr. Don Eagles, executive vice-president of Blue Shield, also discussed the history, progress, and future of the Blue shield plan. Dr. R. W. Pierson was unanimously elected to membership in the Sixth District Medical Society. The society also approved the adoption of plan C for Blue Shield. It was moved and passed that the medical payments of such a plan be based on the relative value fee schedule. At this same session, a move was made and approved to increase district dues by \$10 per year. The custom of sending \$15 to the Sixth District Medical Auxiliary to help defray the expenses of the AAPS essay contest is to be continued.

The next meeting was held on February 24, 1959. On the program was Dr. Irwin Kaiser, associate professor of obstetrics and gynecology at the University of Minnesota Hospitals, who discussed "The Early Detection and Treatment of Carcinoma of the Cervix." Dr. Harris D. Hanson was unanimously elected to membership in the society. Drs. M. S. Jacobson and Marlin Johnson were reelected to the North Dakota Physicians Board of Directors and the Sixth District passed a resolution opposing the milk bill (House bill No. 597).

The next meeting was held on April 2, 1959. The scientific program was presented by the Cancer Caravan under the direction of Dr. C. M. Lund of Williston. Dr. Edgar Harrison, surgical pathologist at the Mayo Clinic, discussed "Exfoliative Cytology," and Dr. M. A. Adson, general surgeon at the Mayo Clinic, discussed "Examination of the Lower Bowel." Approval was given for showing the film entitled "Time and Two Women," and it was the consensus of the committee appointed to review this film that it should be shown to the lay public. No instructions were given to the delegates for the annual convention.

The next regular meeting was on September 22, 1959. The treasurer's report at that time showed a balance of \$600.11. The speaker of the evening was Dr. J. Gordon Borrow of Atlanta, Georgia, who discussed the problem of rheumatic heart disease and preventative medicine and what the Public Health Service was doing about it in Georgia.

The next regular meeting was held on November 16, 1959. This was a special meeting called to discuss the political situation in North Dakota and its relationship

to medicine. Guest of the evening was Dr. J. C. Fawcett, president of the North Dakota State Medical Association, who discussed the advancement of socialized medicine with special reference to the Forand bill. Dr. C. H. Peters, Sixth District councillor, discussed what could be done by each physician in the bipartisan field of politics. A motion was made and passed that a bipartisan legislative committee of 5 or more be appointed by the president to serve actively on legislative activities for this district. It was also advised and passed that a Committee on Aging be appointed to create a positive approach with new ideas and to disseminate these ideas both to the public and the physicians of this society. Resolutions regarding our opposition to the Forand bill were passed to send to each of our representatives and senators. Breakfast meetings were set up with Representative Donald Short and Representative Quentin Burdick to discuss our views on legislation with them. It is felt that these meetings were quite successful in introducing medicine's ideals and medicine's philosophy of free enterprise and maintenance of our form of government.

The next meeting was held on December 14, 1959. Dr. Rudy Froeschle of Hazen was announced chairman of the Bipartisan Legislative Committee, and members are Drs. Philip Dahl, Robert Tudor, Joseph Cleary, and M. S. Jacobson. At this meeting, Drs. W. D. Waldschmidt, J. R. Morton, Hendrika Van Drunen, Arthur Ewert, and Edward Goodman were unanimously elected to membership in the society. The election of officers then took place and are as follows: Dr. Marlin Johnson, Bismarck, president; Dr. Rudy Froeschle, Hazen, vice-president; and Dr. C. R. Montz, Bismarck, reelected secretary-treasurer. Dr. George Lipp of Bismarck was elected to the Board of Censors for a term ending in 1962. Drs. Robert Tudor and Milton Nugent of Bismarck were elected to the House of Delegates for a term ending in 1962, and Dr. Edmund Vinje of Hazen was elected to the Board of Directors of the Blue Shield Corporation for a term ending in 1962. The scientific program of the evening consisted of a film on "Radiation Hazards."

The next meeting was held on February 9, 1960. The scientific program was given by Dr. E. R. Alexander, chief of Communicable Disease Center, Atlanta, Georgia, who discussed "Staphylococcal Disease Infections and Their Control." The Committee on Aging was appointed, with Dr. M. M. Heffron, chairman. Other members are Drs. P. R. Gregware, O. V. Lindelow, and M.A.K. Lommen. Our legislative contacts were discussed further by Dr. Rudy Froeschle, chairman of the Bipartisan Legislative Committee. An additional contribution of \$25 was made to the AAPS, which is being sponsored by the Sixth District Medical Society Auxiliary. Dr. Edwin Boerth reported to the society on the national meeting of Blue Shield and Blue Cross held in Chicago.

The last meeting was held on March 15, 1960, with Mr. Floyd Upham, state safety director, as speaker.

Membership in the district is as follows: active members, 75; retired members, 1; honorary members, 7; and limited members, 1. Eight new members joined the society. Members who transferred were Dr. John Anthony to the Northwest District and Dr. E. Bryant to Minnesota.

Members of the House of Delegates are as follows: Dr. C. J. Baumgartner, term expiring in 1960; Dr. E. Vinje, term expiring in 1960; Dr. M. A. K. Lommen, term expiring in 1961; Dr. M. E. Nugent, term expiring in 1962; and Dr. R. B. Tudor, term expiring in 1962.

Attendance at meetings has been excellent, usually averaging 45 to 55 members at each session.

C. H. PETERS, M.D., Councillor

Seventh District

During the past year, 8 dinner meetings were held.

The first meeting was held on April 3, 1959, with 19 members present. Guests were Dr. Carroll Lund, co-ordinator of the Cancer Caravan; Lyle Limond, executive secretary of the North Dakota State Medical Association; Dr. Dave Halliday of Kenmare; and Drs. Sakai, Thiery, Mishchenko, Icenogle, and Laurich of the State Hospital staff. The program was provided by Dr. Lund and his feats of magic, following which Dr. Martin Adson, surgeon at the Mayo Clinic, spoke upon "Carcinoma of the Colon and Rectum," and Dr. Edgar Harrison, surgical pathologist at the Mayo Clinic, gave an excellent résumé on "Exfoliative Cytology."

The second meeting was held on April 23, 1959, and 13 members were present. There was a short business meeting after which a cancer film "Time and Two Women" was shown.

The third meeting was held on May 21, 1959, and 19 members were present. Guests were Dr. Tom Rulon of the Mayo Clinic, Dr. Icenogle of the State Hospital staff, and Delbert Ilvinko and Richard Truax, medical students from the University of North Dakota, here under preceptorships. Drs. Pederson, Van der Linde, and Nierling reported on the business of the state medical association meeting held at Bismarck earlier in the month. A program was presented by Dr. William G. Hemenway, associate professor of Otolaryngology at the University of Chicago, who spoke on the subject of "Diseases of the Parotid Gland," and dealt briefly with treatment and reconstruction after radical surgery.

The next meeting, which was our business and organizational meeting of the year, was held on December 10, 1959. There were 17 members in attendance. The following guests actually presented the program: Dr. John Fawcett of Devils Lake, president of the North Dakota State Medical Association, spoke about the overall medical and political scene in North Dakota and in the United States; Dr. Clifford Peters of Bismarck, chairman of the Medical Economics Committee and co-chairman of the Legislative Committee, spoke at length on problems related to the Welfare Board and medical fees, to National Legislation, and to Blue Cross and Blue Shield and on dangers of the Forand bill. Mr. Lyle Limond, executive secretary of the North Dakota State Medical Association, and Don Eagles, executive secretary of the Blue Shield, spoke about problems in their particular areas. Three guests from the State Hospital also attended the meeting.

Our next meeting was held on December 17, 1959, with 18 members in attendance. Guests were Dr. Thompson, an intern at St. Luke's Hospital in Fargo, Dr. Jensen of Valley City, and Dr. Rioux of the State Hospital. Dr. Mark Traynor of the Fargo Clinic presented a paper on the "Unusual causes of Upper Gastrointestinal Bleeding." This was followed by a short organizational meeting, at which time a Legislative Committee was appointed with Dr. Elsworth as chairman.

The next meeting was on January 26, 1960, at which time 16 members were present. Six guests were present. The program was presented by Dr. Perry Triggs of the Fargo Clinic, who spoke on "Hemorrhagic Diseases."

The next meeting was held at the Elks Club on February 25, 1960. There were 18 members in attendance, and 3 guests were present. The program consisted of a report of the activities of the Legislative Committee during the preceding months. The scientific portion of the program was presented by Dr. Edgar Hannz of the Grand Forks Clinic, who spoke on "Diabetes Mellitus."

This was an excellent presentation and again demonstrated how worthwhile programs presented by state society members are. The meeting was closed with a film "Tonsillectomy Techniques."

The next regular meeting was held on March 17, 1960, at the Elks Club. There were 21 members and 6 guests present. The program was presented by Dr. Fleteher Miller, associate professor of surgery at the University of Minnesota, who spoke on "Pediatric Surgery and Vascular Reconstructive Surgery."

Officers for 1960 are: president, Dr. John A. Swenson, Jamestown; vice-president, Dr. Clarence Martin, Kensal; and secretary-treasurer, Dr. R. D. Nierling, Jamestown. Delegates: Drs. John N. Elsworth, Jamestown, and John Van der Linde, Jamestown. Alternate delegates: Drs. John A. Swenson, Jamestown, and Robert E. Lucy, Jamestown. Blue Shield directors: Drs. Joseph Sorkness, Jamestown, and E. J. Larson, Jamestown. Blue Cross representatives: Drs. Joseph Sorkness, Jamestown; John Van der Linde, Jamestown; R. L. McFadden, Jamestown; Ellis Oster, Ellendale; and J. N. Elsworth, Jamestown.

Membership of the district includes 31 active members and 1 retired member. The following members transferred out of state: Drs. John Freeman to Omaha; John Young to Traverse City, Michigan; and Kenneth Harris to Great Britain. One member, Dr. P. G. Arzt, Jamestown, died during the year.

T. E. PEDERSON, M.D., Counsellor

Eighth District

The Eighth District Medical Society is comprised of physicians practicing in Williston, Crosby, Watford City, and Tioga. At present, we have 17 members.

Five meetings were held during the past year.

On January 28, 1959, the annual business meeting was held, and the following officers were elected: president, Dr. Duane Pile; vice-president, Dr. Chester Bornd; and secretary-treasurer, Dr. John Keller. Delegate: Dr. Dean Strinden. Alternate delegate: Dr. E. J. Hagan. Representatives to North Dakota Physicians' Service: Drs. J. D. Craven and D. E. Skjei.

On March 28, 1959, a dinner meeting was held at the Williston Elks Club followed by a Cancer Caravan program. Dr. Harrison of the Mayo Clinic gave an interesting talk on "Exfoliative Cytology" and illustrated his points with slides. Dr. Martin Adson of the Mayo Clinic then discussed "Cancer of the Colon and Rectum." The meeting was well attended.

On September 28, 1959, a dinner meeting was held at the Moose Hall in Crosby. Dr. Walter Pretorius of the United States Public Health Service spoke on "Primary Rheumatic Fever Prophylaxis." This was a report of a study carried on in the Bismarck-Mandan area. Dr. Peterson of the State Health Department then spoke about the establishment of a poison control center for the Williston area.

On November 18, 1959, a dinner meeting was held at the Plainsman Hotel, which was attended by our association president, Dr. John Fawcett, and Dr. O. W. Johnson, head of the association Legislative Committee.

On January 27, 1960, a dinner meeting was held at the Plainsman Hotel. This was our annual business meeting. The following officers were elected: president, Dr. Chester Bornd; vice-president, Dr. H. Charles Walker, Jr.; and secretary-treasurer, Dr. John Keller. Delegate: Dr. Dean Strinden. Alternate delegate: Dr. E. J. Hagan. Representatives to North Dakota Physicians' Service: Drs. J. D. Craven and D. E. Skjei. Board of Censors: Drs.

W. A. Wright, chairman, and Chester Borrud, J. D. Craven, and Duane Pile.

J. D. CRAVEN, M.D., Councillor

Ninth District

The Ninth District, also known as the Southwestern District Medical Society held 6 official meetings during the calendar year of 1959. We have 29 members, 3 of whom are retired and 1 of whom is practicing in California.

The first meeting was held February 14, 1959, at the Ray Hotel, Dickinson. Duties of the delegates and alternate delegates to the state association were discussed. We formulated a telegram to be sent to the state legislature showing our disapproval of a chiropractic bill under consideration. A sum of \$25 was given to the medical auxiliary for a prize in the medical education essay contest. The scientific aspect of the meeting was held at St. Joseph's Hospital and concerned civil defense measures and a film on "Malignancy in Women."

The second meeting was held April 1, 1959, at the Ray Hotel. This was essentially a scientific meeting, with a majority of the program being presented by Dr. Edgar G. Harrison of the Mayo Clinic, who spoke on "Exfoliative Cytology." Dr. Martin A. Adson, also of the Mayo Clinic, spoke on "Cancer of the Colon and Rectum." These speakers were furnished by the North Dakota Cancer Society.

The third meeting was held June 13, 1959, at Mott. We were guests of Dr. Robert E. Hankins of Mott. There was no scientific presentation at this meeting. The disturbed situation at the Jamestown State Hospital was discussed, but no official action was taken.

The fourth meeting was held October 10, 1959, at the Ray Hotel. The 2 members of the board of directors of Blue Shield for this district discussed the present status of the Blue Shield plan and contemplated changes in rates. There was also a discussion of the overuse of Blue Cross and Blue Shield. A scientific meeting was then held at St. Joseph's Hospital. Two films were shown: "Premarin and Blood Coagulation" and "Epilepsy."

The fifth meeting was held November 17, 1959, at the Palm Beach Club, Dickinson. This was entirely a business meeting. A communication was read from Merck Sharp & Dohme, which stated their plan of furnishing guest speakers for district society meetings. Another communication from the American Medical Association concerning material for TV programs was read. This was followed by a rather long discussion. A legislative committee composed of 6 members was appointed by our president, Dr. Richard F. Raasch.

We were honored that evening by having with us the president of the state association, Dr. John Fawcett, and Dr. C. H. Peters, chairman of the Medical Economics committee. The Forand bill was thoroughly discussed, and this society formally voted to use all its power to oppose this suggested legislation. Dr. Peters spoke at length on the State Welfare Board fee schedule and the extensive negotiations with the North Dakota State Welfare Board. He stated that final approval had not yet been given by the State Welfare Board but that definite advancements had been made by the state association negotiating team.

The sixth and final meeting was held December 12, 1959, at the Ray Hotel. The medical auxiliary requested \$50 from the district society for medical essay contest, and this was granted. Our guest speaker for the scientific meeting was Dr. William Walton of Billings, Montana, an orthopedist, who discussed "Acute Backache."

The nominating committee presented the following list of district society officers for approval and they were unanimously accepted. Officers elected for the year 1960 were: president, Dr. Walter C. Hancwald, Richardton; vice-president, Dr. Norman B. Ordahl, Dickinson; and secretary, Dr. Donald J. Reichert, Dickinson. Delegates: Drs. Keith G. Foster, Dickinson, and Robert F. Gilliland, Dickinson. Alternate delegates: Drs. Robert E. Hankins, Mott, and Henry Slominski, Richardton. Councillors: Drs. James Knickerbocker, Hettinger; Robert C. Thom, Bowman; and William M. Buckingham, Elgin. North Dakota Physician Corporation members: Drs. Robert W. Rodgers, Amos R. Gilsdorf, Robert F. Gilliland, and Keith G. Foster, all of Dickinson. North Dakota Physician Service Board of Directors: Drs. Robert W. Rodgers and Amos R. Gilsdorf, both of Dickinson.

During the year of 1959, no urgent medical emergencies arose in our society.

Two new members were added during the year: Drs. Paul A. Ahlness, Bowman; and Walter S. Skwarok, Hebron. Two members transferred out of the state: Drs. Gladys E. Martin, Dickinson; and Julian Tosky, Hebron. None of the members transferred to other district societies; no deaths occurred; and no physicians reside and practice in the district who are not members.

A. R. GILSDORF, M.D., Councillor

Tenth District

The Tenth District Medical Society held 6 meetings in 1959.

On January 9, we had discussion of group malpractice insurance and a fairly lengthy discussion concerning Blue Shield.

A taped lecture by Dr. Thorek on "Acute Appendicitis" was used as the scientific part of the program for March eighteenth.

The Grand Rounds film on "Chest Disease" was shown May 29. At this time, the State Hospital situation was discussed.

On July 22, a business meeting was held.

Our meeting on October 14 centered around the Grand Rounds film, "Open Heart Surgery."

December 9 found us watching a film on "Current Trends on Diabetes." At this time, the following officers were elected for 1960: president, Dr. James M. Little, Mayville; vice-president, Dr. D. N. Mergens, Hillsboro; and secretary-treasurer, Dr. R. W. McLean, Hillsboro. Delegate: Dr. R. W. McLean, Hillsboro. Alternate delegate: Dr. James M. Little, Mayville. Censors: Drs. H. A. LaFleur, three years; Mervin Rosenberg, two years; and R. C. Little, one year.

The membership consists of 10 active members. Dr. Pearson transferred to the Sixth District. Dr. Wakefield transferred from the Fifth District to this district. Dr. A. J. Kjelland died in January 1960. No non-members are practicing in this district.

K. G. VANDERCON, M.D., Councillor

REPORTS OF STANDING COMMITTEES

Committee on Medical Education

A meeting of this committee was held Sunday, May 3, 1959, at 8:30 A.M. in the Petroleum Room at the Prince Hotel, Bismarck. All members of the committee, except 2, were present. Dean Harwood reported that all the students who are completing their sophomore year have been accepted in other medical schools for their last two years.

Dean Harwood stated that the applicants for the freshman class next fall averaged higher grades in their pre-

medical training than in the previous classes. At this time, it appears that there are sufficient applicants from North Dakota to fill the class, so that it is questionable whether they will be able to take any students from outside of the state in the freshman medical class next fall.

He felt that all the doctors in the state should encourage all good students who are interested in medicine to go into medicine and to apply at North Dakota.

The members of the committee who attended the postgraduate course, which was put on by the University and the Academy of General Practice felt that it had been very well done and moved that the dean and the staff at the medical school be complimented on the excellence of this program. The members of the committee felt that the medical school should offer a postgraduate course each year.

One doctor who has taken advantage of the loan fund will return to the state this year, and 7 will return next year. The dean thought that these men would all return to the state to practice instead of paying off their loan.

The preceptorship system for the sophomore students was discussed. The program apparently has been satisfactory, but it seemed to be the opinion of the committee, including Dean Harwood, that the program might be further improved if Dean Harwood could visit the various centers of training when the students are there. Dean Harwood told the committee that he would try this year to visit the majority of these centers.

The \$2,000 fellowship for a medical student set up by the National Foundation was discussed. It was decided that the student for this fellowship should be selected by Dean Harwood, Arnie Arneson, and their associated committees at the medical school.

None of the members of the committee felt that it would be necessary for this committee to meet during the coming year unless some special situation developed.

On May 8, the following resolution was received from Mr. Limond:

RESOLUTION

Whereas, at the present time, the State of North Dakota does not have a toxicological laboratory, or a qualified toxicologist, and *Whereas*, the only work of this type is presently done by Dr. G. A. Abbott, who is 80 years old and retired, and

Whereas, the present situation does not make it possible to have a full-time toxicologist in the state because of the salary involved, and

Whereas, adequate laboratory facilities are now present at the University of North Dakota Medical School and would not require duplication, and

Whereas, it could be possible for one of the present laboratory staff to obtain this training in lieu of the difficulty presented in obtaining a fully qualified toxicologist,

Now, therefore, be it resolved that the Committee on Medical Education be directed to investigate the possibility of establishing an adequate toxicological laboratory and its implementation through the procurement or training of a qualified toxicologist.

This resolution was forwarded to Dean Harwood. On December 1, 1959, he reported as follows:

"We are working on the toxicologist problem. We have, of course, all of the equipment that anyone might need to carry on any tests that would need to be done. I have written Dr. Alan Moritz, a world famous toxicologist, to see if there are any formal or informal programs to which we might send someone for special training. At the present time, there is no one in Dr. Cornatzer's Department of Biochemistry who has the time to spend at it. As you realize, our problem is one that if we set up a toxicology program, someone must do a great deal of detailed and time-consuming analysis. The volume will be small enough so that it would not seem feasible to hire and train a technician. We will have to find someone

who is willing to devote the time when the occasion arises to do these tests."

They have not been able to find a solution to this problem up to this time.

We tried to have a meeting of this committee during the winter months. Three possible meetings were set up, but we were unable to obtain a quorum for any of the dates selected. The idea of having an interim meeting was, therefore, abandoned for this year.

Dr. Harwood's report on the Medical School is as follows:

"Our chief problem this year, a fairly alarming one, and one which might take up this entire report, is the diminishing number of qualified North Dakota students applying to the medical school. Last year, we had an excellent year. We admitted 44 North Dakota students, although 2 withdrew before school actually started in September. We filled these places with 2 nonresident students.

"By comparison, the situation this year with respect to North Dakota applicants is alarming. We have only 37 compared to 56 last year. To date we have accepted 23, rejected 3, lost 1 to another medical school, and 1 has withdrawn. We have deferred action on 8, and acceptances from this group will depend on second semester grades. One applicant has not been interviewed. We do have an adequate supply of nonresident applicants.

"This dearth of qualified applicants is general. Countrywide, nearly each year there is a drop in applicants, some of whom are poor students and some grossly inadequately prepared.

	Applicants	First-year places	Applicants per place
1949	24,434	6,688	3.6:1
1951	19,920	7,436	2.7:1
1954	14,538	7,576	1.9:1
1958	15,170	8,030	

"There is a great deal of interest throughout the nation in this problem of the dropping off of well-qualified students. As many causes are suggested as there are causes for the Civil War. Among them is the competition we are getting from engineering in this age of the sputnik. Graduate schools have been expanding, and a senior student in college is faced with a choice of going into graduate work in physics, mathematics, chemistry, or biology with a *stipend* of \$1,500 a year or entering medical school at a *cost* of \$1,200 a year. This differential is considerable.

"Our University, including the medical school, is handling an increased number of graduate students, many under federal programs, to increase the number of the teachers qualified to teach in college. We are doing our share in preparing for the projected increase in college enrollment and increased need for trained teachers.

"Our state president recently remarked about the trend of socialized medicine deterring students from applying, and there have been comments from other sources about the loss of prestige of the doctor, the increasing number of malpractice suits, and so forth. All of these causes are part of the picture, I am sure. However, I think that we do have a reasonable number of students who are interested in medicine but who fail to properly prepare themselves or who drop out in college.

"Our first-year medical students are close to this problem, since a short time ago they were part of this group which concerns us. They were impressed that in their first year of college there were numerous engineers and

premedical students in their class. Many did poorly or failed in chemistry or mathematics and changed to other programs. They felt that these students had failed to take the courses of mathematics and science in high school and were lost in college. They feel the defect is intellectual training rather than motivation. Interestingly enough, none of this group had considered the threat of socialized medicine a factor in their choice of career. I volunteered that the cooperation of the physicians in support of present plans of providing medical care through voluntary and private agencies is, and will be, the greatest deterrent against socialized medicine.

"The first student who borrowed from the Medical Center Loan Fund returned to Rolla to practice, and this year there will be 5 more. We have loaned a total of \$183,000 to such North Dakota students; that is, students in their third and fourth year. Repayments are beginning to be made.

"There are no essential changes in our faculty or in our transfer situation.

"The Ireland laboratories are in operation, and we are selecting a Hill research professor, supported by a five-year grant of \$75,000 from the Hill Foundation in Minneapolis.

"Again we appreciate the support of the physicians of the state in their important contribution of instruction in clinical clerkships. We have 4 sons of physicians enrolled in medical school, all of whom, we are happy to say, are doing well.

"We are considering the need for more space, because our graduate program is growing and research areas are becoming filled.

"May we close with a plea that you physicians in your home town try to emphasize to your school boards and students the importance of starting a good foundation in mathematics and the sciences. We will try to do our part after the students get here."

Dean Harwood informs us that a postgraduate course will be held next fall on the Thursday preceding the meeting of the Academy of General Practice in Grand Forks.

The meeting of this committee this year will be held at noon on Saturday, April 30, 1960.

If anything comes before this committee that should be acted on by the House of Delegates or the Council, it can be submitted to them immediately after our meeting, since they will still be in session.

H. M. BERG, M.D., Chairman

Committee on Necrology and Medical History

It is true that we shall not be able to reach perfection, but in our struggle toward it we shall strengthen our characters and give stability to our ideas, so that, whilst ever advancing calmly in the same direction, we shall be rendered capable of applying the faculties with which we have been gifted to the best possible account.

CONFUCIUS

JOHN S. McARDLE, M.D.

Dr. John Stephen McArdle was killed in an Automobile accident May 29, 1959, at the age of 42. He was born September 16, 1916, at Winnipeg, Manitoba. He prepared for the medical profession at the University of Manitoba Faculty of Medicine in Winnipeg from which he graduated in 1952. He served his internship at St. Boniface Hospital.

He entered the Canadian Air Force in 1933 and was discharged on September 4, 1939.

He was married on June 27, 1947, and moved to Minot

April 8, 1953. He had practiced medicine at the Great Plains Clinic in Minot.

He was a member of the First Presbyterian church and the Minot Lions Club. Dr. McArdle and his wife became naturalized citizens of the United States only one day prior to his death.

He was active in the Ward County chapters of the National Foundation, Multiple Sclerosis, and the Heart Association.

Surviving are his wife and 4 children.

ARCHIE D. McCANNEL, M.D.

Dr. Archie D. McCannel, Minot physician and civic leader for more than a half century, died July 22, 1959. He had undergone major surgery four days prior to his death. Dr. McCannel, 80, had been in poor health since he narrowly escaped drowning when trapped in his automobile as it plunged into the Mouse River west of Minot in 1956.

During his youth, he thought he would like to be a druggist and, in 1901, settled in the small town of Richberg, North Dakota, near the present site of Westhope, where he opened his own drug store. After about a year, he decided instead to become a physician and returned to Canada to study medicine.

He received his early education in Ontario, where he was born. He was graduated from the University of Toronto Faculty of Medicine in 1906 and from there went to London to study, specializing in eye, ear, nose, and throat work.

After his postgraduate study in London, he went to Minot in 1907. He operated the McCannel Clinic in Minot for twenty years and received recognition for both his medical and fraternal activities. He was president of the North Dakota Medical Association in 1936 and was a life member of the American College of Surgeons. He was made a member of the 50-Year Club and an honorary member of the North Dakota State Medical Association in 1956.

He was a past governor of the Ninth Rotary district and was past grand commander of the North Dakota Knights Templars. Dr. McCannel was a former member of the Public Welfare Board of North Dakota and, in 1943, was appointed to the State Board of Higher Education. He was also a member of the Scottish Rite Bodies in Minot. In September 1947, he retired as chairman of the Ward County chapter of the Red Cross after serving for thirty years.

He was long active in the First Presbyterian Church of Minot. He served as superintendent of the Sunday school for thirty years. In 1949, he was appointed to the board of directors of the Lutheran Home for the Aged at Minot. He was past president of the Great Plains area council and was active in Boy Scout work.

From October 31, 1940, until January 14, 1947, he served as president of the First National Bank of Minot.

Dr. Willard Wright paid tribute to Dr. McCannel in his excellent article in the October 1959 issue of THE JOURNAL-LANCET.

Dr. McCannel married Violet Rose of London, Ontario, in 1908. She and 2 daughters and 2 sons survive.

PHILIP G. ARZT, M.D.

Dr. Philip G. Arzt, 78, physician and surgeon in Jamestown for more than fifty years, died January 14, 1960. He had been in failing health since fracturing a hip in 1959.

He was born in St. Paul, August 19, 1881, and attended the St. Paul public schools and Baldwin Seminary. He was graduated from the University of Minnesota

Medical School in 1905 and was licensed to practice medicine in North Dakota in 1906. He also was licensed in Minnesota. He practiced medicine for one year at Fingal, North Dakota, and went to Jamestown in 1908.

He was chief of staff of Trinity Hospital for many years and helped organize a medical clinic. He was a director and vice-president of the Jamestown National Bank, secretary of Gladstone Hotel Company, and a member of the Jamestown Park Board since 1931 and its president since 1948. He was a member of the State Board of Medical Examiners for many years and a fellow and life member in the American College of Surgeons. He was president of the North Dakota State Medical Association in 1948. In 1955, he became a member of the 50-Year Club of the association and, in 1956, became an honorary member.

Dr. Arzt also was a charter member and past district governor of Kiwanis and a member of the Masonic Lodge.

He is survived by his wife, 2 sons, and 3 daughters.

ANDREW ARTHUR KJELLAND, M.D.

Dr. Andrew A. Kjelland, 79, a physician in Hatton, North Dakota, since 1911, died January 18, 1960, in an area hospital. Dr. Kjelland had suffered a cerebral hemorrhage in May 1957 and had been a hospital patient since.

Born at Rushford, Minnesota, July 30, 1880, Dr. Kjelland was the son of Mr. and Mrs. Ole A. Kjelland. He attended grade school in Rushford, high school in Black River Falls, Wisconsin, and was graduated from the University of Minnesota Medical School in 1910.

After a year as intern in Luther Hospital, St. Paul, he began practice in Hatton. In 1911, he became a staff member of the Deaconess Hospital at Northwood. In 1948, he was honored for services to the Hatton area. He married Blanche Tucker in Minneapolis August 5, 1911. Mrs. Kjelland died on November 16, 1955.

Dr. Kjelland is survived by a daughter, a brother, 2 sisters, and 3 grandchildren.

FRANK E. WHEELON, M.D.

Dr. Frank E. Wheelon, 82, retired Minot physician, died in Lake City, Florida, February 23, 1960, the day before his 83rd birthday.

Born in Wheaton, Illinois, February 24, 1877, Dr. Wheelon came to Dakota Territory in 1881 with his parents, who settled at Cummings. He attended the University of North Dakota and received his medical degree at the University of Minnesota in 1899.

After a year at Welonic, Minnesota, he went to North Dakota and practiced at Fingal, Esmond, and Maddock until 1909 when he settled in Minot. He served in the Army Medical Corps in World War I and was overseas fourteen months.

He was in private practice in Minot for thirty-seven years until his appointment as director of the First District Health Unit in Minot in 1946. He served as director until his retirement in June 1957, doubling as Ward county health officer.

Following his retirement, he and Mrs. Wheelon lived in Fresno, California, for six months and then moved to Lanark Village, Florida.

Dr. Wheelon was a member during his entire residency in Minot in the Inquirers Club and was a member of the American Legion, Elks Lodge, and the Last Man's Club, an organization of World War I veterans. He was made a 50-Year Club member of the North Dakota State

Medical Association in 1950 and became an honorary member in 1951.

He is survived by his wife, 2 sons, and 2 daughters.

OSCAR T. BENSON, M.D.

Dr. Oscar T. Benson, physician at Glen Ullin, North Dakota, for more than forty years and a brother of former Minnesota Governor Elmer Benson, died in Hollywood, California, where he had lived in recent years, on March 7, 1960, at the age of 80.

He was born November 30, 1879. In 1905, he received his medical degree from the University of Minnesota and was licensed to practice medicine in North Dakota in January 1907. He practiced in Glen Ullin for over forty years and was a member of the Sixth District Medical Society.

In 1938, Dr. Benson was honored by the community of Glen Ullin for his long service to the area.

He was president of the Glen Ullin school board for almost twenty years and was active in Boy Scout work. He had been a member of the El Zagal Shrine Temple at Fargo since 1910.

In 1955, he was honored by membership in the 50-Year Club of the North Dakota State Medical Association and became an honorary member in 1957.

Survivors include his wife, Helen, and 2 sons and 2 sisters.

DONALD L. OLSON, M.D.

Dr. Donald L. Olson, 39, orthopedic surgeon in Fargo, died December 13, 1959, as the result of injuries sustained in an automobile accident the previous day.

Born in Minneapolis on March 7, 1920, Dr. Olson received both his medical degree and his degree in orthopedic surgery there from the University of Minnesota.

Before establishing practice in Fargo, he was on the staff of the Gillette State Hospital for Crippled Children, St. Paul, and the Shriners Hospital for Crippled Children, Minneapolis, and was chief of the orthopedic section of the Minneapolis Veterans Hospital.

He served three years as a Navy fighter pilot in World War II and had continued his interest in flying, being a member of the Flying Physicians Association and frequently flying on vacation and professional trips. He also was a member of the Civil Air Patrol.

Besides his private practice, Dr. Olson was on the staff of St. John's and St. Augar hospitals and was a consultant at the Fargo Veterans Hospital. He was a member of the First District Medical Society, the North Dakota State Medical Association, and the American Medical Association. His practice in Fargo was formerly that of Dr. Harry J. Fortin, a Fargo orthopedic surgeon for many years.

Surviving Dr. Olson are his wife, 3 sons, and 3 daughters.

J. L. DEVINE, SR., M.D.

Dr. J. L. Devine, Sr., 79, pioneer resident of North Dakota who practiced medicine in the state for more than fifty years, died April 1, 1960. He had been in failing health for several years.

Born on February 8, 1881, in Dexter, Michigan, Dr. Devine came with his family to Devils Lake in the 80's at a time when the railroad ended there. After completing his education, and graduating from the University of Minnesota Medical School in 1901, he was licensed to practice medicine in North Dakota the same year. He began his medical practice at Birlford, North Dakota, in 1901 where he remained for one year. In 1905, he moved

to Lansford, North Dakota, where he practiced for ten years before moving to Minot in 1915 where he resided until the time of his death.

During his entire medical career in Minot, he was associated with St. Joseph's Hospital and served for a number of years as its chief of staff. He also served as a member of the Nursing School Committee of the hospital and as a member of the Staff Executive Committee. Through his years of practice, he played a major role in improving the teaching of nurses at St. Joseph's. He believed that without good nursing, the finest medical skills were hampered.

Dr. Devine was a member of the Minot Elks Lodge since 1916 and served in 1933 and 1934 as exalted ruler and later as president of the Past Exalted Rulers Association.

During World War II, he served on the Selective Service Board and received a congressional citation for his service.

He was a long-time member of the Northwest District Medical Society, the North Dakota State Medical Association, and the American Medical Association. He was made a member of the 50-Year Club of the state association and became an honorary member in 1954.

Survivors include a son, Dr. J. L. Devine, Jr., Minot, and 3 grandchildren.

E. H. BOERTH, M.D., Co-Chairman

Committee on Legislation

The activity of the Legislative Committee has been considerably greater during these off-legislative years than it has been in years gone by.

The American Medical Association invited the co-chairmen of the Legislative Committee, the executive secretary, and the president of the North Dakota State Medical Association to attend a conference in St. Louis in October 1959, at which time various approaches to the solving of attempted socialization of various segments of our population were discussed, primarily in the nature of the Forand bill. This seminar was very instructive. However, I must state that I came to the conclusion, following a two-day conference, that nothing had been done to offer any alternative plan. This needed to be done in order to forestall such legislation on a federal basis in Washington. As such, it would be necessary to institute some old age program at home in every state in the Union.

Following this conference, your chairman did attend a meeting of the Legislative Committee of the 6 North Central states: namely, Nebraska, Iowa, South Dakota, Minnesota, North Dakota, and Wisconsin. Again, it appeared to your chairman that, although we were faced with a problem, no real alternative plan sufficiently satisfactory to forestall legislation of the Forand-type bill had been presented.

The Legislative Committee met in Fargo and discussed again many of the facets of the type of legislation which the medical profession is faced with on a national basis and, along with various other problems, arrived at no definitive action except for one thing. It was decided that a speaker should approach every society in the state and discuss methods and means of voicing our opposition to the socialized form of legislation which was being promoted. Dr. Cliff Peters of Bismarck was kind enough to take the southern tier of towns in the state, and your chairman covered the northern tier of towns in the state, and, in all, 8 societies met to discuss the Forand bill and its particular type of legislation. Accordingly, a legislative committee has been set up on a local basis in every

society in the state for the purpose of scrutinizing all forms of bills which may come up either on a local or national basis which may involve medicine at large. Your state committee will be very glad to listen to any suggestions from any particular society relative to legislative matters.

The Legislative Committee met again on January 30. Various outside groups were invited to be present at this meeting, and the facets of legislative action on a local, state, and national basis were taken up. However, final action was not very definitive.

It is the opinion of your chairman that the next legislative session is going to be faced again with an increased number of medical bills. At each legislative session, the number of bills has increased by almost 100 per cent, and, if our judgment is not too far off, I believe that it will increase by almost that much in the forthcoming session. For instance, on February 28, 1960, an associated press release indicated that one of our local members of the Senate proposes to throw a bill into the hoppers making a 1 mill levy against the taxpayers of North Dakota for rendering total medical care in the state, which, for all practical purposes, would be socialization of medicine in the state, should such legislation come to pass.

There is little doubt in my mind but that an individual who would introduce such legislation in the state is being prodded from elsewhere. Very few in the state are socially inclined far enough to the left to initiate such legislation on their own.

It now remains for the medical profession in the state at large to be on their toes constantly relative to any changes that may be coming forth in the line of legislation and to lend a hand individually and collectively to fight any legislation that does in any way hamper, harm, or socialize our form of practice.

O. W. JOHNSON, M.D., Chairman

Committee on Public Relations

The PR Institute, sponsored by the AMA, was held in the Ambassador West Hotel in Chicago on August 20 and 21, 1959. It was attended by Dr. J. C. Fawcett, Mr. Limond, and myself. The theme of the conference was "Is Medicine on the Right Track?" The question was posed as to whether to purchase your own ticket or "get a free ride."

Various types of socialized medicine were discussed by the panel, which was composed of organizations of doctors from Germany, Canada, and England. The second question posed was whether legislation was endeavoring to derail the American system of medicine. This was adequately discussed by the press and by legislative experts of the American Medical Association and Washington. The program of Science Fairs was discussed, and there was a very fine panel discussion by career experts in the field of science and electronics. This gave us a very clear insight into what is being done by the American Medical Association as well as the state societies to promote and stimulate interest in our youth.

Also, fadisms, quack medicine, and other numerous ways of deceiving the public were brought out. The methods utilized by the medical societies on various levels, the Food and Drug Administration, and the press to combat these illicit facets were discussed.

Medical costs, especially with a view to the thinking in Washington, was brought out in the topic, "Is the Cost of Medicine Too High?" Outstanding speakers were on hand who spoke especially in reference to the C.I.O., U.W.A., insurance companies, and, last but not least,

the Forand bill. Adequate means and methods were outlined for combating the legislation as devised by Forand, and it appears that the cooperation of various health associations would more than adequately create successful lobbying in Washington if the doctors in their grass roots would participate.

We were also given a look into the future as to what our forthcoming problems will be and the ways and means of solving same by radio, TV, press relations, and field services offered by the AMA in the fields of aging, school help, and legislation favoring medicine. With this Institute forming a background, an adequate program was discussed and has been instituted by our executive secretary, especially with reference to the Forand bill.

If everything as outlined goes well, medicine should be triumphant.

JOHN T. CARTWRIGHT, M.D., Chairman

Committee on Official Publication

The Committee on Official Publication held one meeting during the year.

The committee recommends that THE JOURNAL-LANCET be designated as the official publication for the North Dakota State Medical Association for another three-year period.

Since THE JOURNAL-LANCET is the official publication for the North Dakota State Medical Association, it is recommended that some recognition of this fact be given on the cover of its monthly publication.

E. H. BOERTEL, M.D., Chairman

Committee on Public Health

No formal meeting of this committee was held during the past year. However, at the present time, we are polling the members to try to come up with some method of evaluating the efficiency of the nursing care in the 19 nursing homes in North Dakota. This service was requested by the Public Welfare Administration after consultation with the Advisory Committee to the Public Welfare Board, of which Dr. M. E. Nugent of Bismarck is chairman. We hope to come up with some recommendations in the first part of April.

The chairman of this committee was invited by the North Dakota State Health Department to attend the symposium on venereal diseases last April. It was held in St. Louis, under the direction of the United States Public Health Service. It was a very interesting meeting, and it is worthy of note that the venereal disease rate is increasing in the state of North Dakota, a large proportion of it being among persons of teen age.

I was also privileged, with the cooperation of the City of Bismarck and the North Dakota State Health Department, to attend a week's course in epidemiology conducted at the Communicable Disease Center at Atlanta. This was a very interesting course, and I would highly recommend it to anyone who is interested in public health and in epidemiology particularly.

Last May, I was also privileged to attend the meeting of the Heart Disease Control section of the United States Public Health Service. I listened to the paper by Dr. Walter Pretorius, who reported on the work that had been done in Bismarck on rheumatic fever. As you know, Bismarck has had the pleasure of cooperating with the North Dakota State Health Department and the United States Public Health Service in carrying out a rheumatic fever control program under the direction of Dr. Walter Pretorius and, during the past year, Dr. Channing Nicholas. This paper was very well received, and the men in charge expressed the hope that we would be able

to continue this program for some time. This, I believe, will be done.

As the legislature meets in 1961, we in public health here in Bismarck feel sure that some kind of a milk bill will be introduced. This has never failed for fifteen or twenty years, and I have no reason to feel that 1961 will be any different. It is my hope that the members of the North Dakota State Medical Association will acquaint themselves with such bills when introduced and that we may plan on their cooperation in helping to keep milk sanitation at a very high level.

PERCY L. OWENS, M.D., Chairman

Committee on Medical Economics

This committee has been active over the past year in many different fields. In June 1959, a new agreement was reached with the Workmen's Compensation Board, which resulted in an increase in fees of a substantial amount in most categories. The committee had endeavored to promote and educate the Workmen's Compensation Board into the merits and value of the relative value fee schedule. Up to this time, we have not been successful, primarily because the relative fee schedule has not yet been put into effect by Blue Shield. We believe that once this schedule is widely used in North Dakota, the Workmen's Compensation Board will see the merit and value of such a program in their department. At that time, further negotiations for a conversion factor should be made with them.

Continued efforts and negotiations were made with the Public Welfare Board of North Dakota throughout 1959 and through January 1960, finally resulting in a mutually agreed upon new fee schedule for welfare recipients, which became effective February 1, 1960. This schedule was adopted only after numerous meetings with the Public Welfare Board staff and after 3 separate meetings with the Public Welfare Board itself, 2 of which were attended by the governor of North Dakota. Again, we attempted to promote, educate, and emphasize the value of the relative value approach. Again, we were unsuccessful, as the board felt that this is a new and untried area and it was unwilling to adopt this program.

The following advances, however, were adopted. The schedule for the eye, ear, nose, and throat field was readjusted, bringing it up to a level of other procedures in the old schedule. At that point, a 15 per cent increase across the board of all procedures was agreed upon. However, house calls during the day went up from \$3 to \$4 and, during the night, from \$5 to \$6. Office calls went up from \$2 to \$2.50 and hospital calls for acute cases changed from \$2 per day to a \$4, \$3, and \$2 schedule. These last 3 items did not follow the 15 per cent increase across the board, but actually amounted to much more than this amount. In addition, open reductions for fractures were boosted 50 per cent over closed reductions.

Perhaps the greatest contribution of this new schedule was the agreement and understanding of various controversial problems affecting the practice of medicine between Public Welfare Board recipients, the County Welfare Board, the State Public Welfare Board, and members of the State Medical Association. Many areas of disagreement exist, and these were spelled out in a definitive fashion, so that misunderstanding should not occur in these areas in the future. In addition, another advance was made with the appointment of a committee of 8 physicians throughout the state to act as an Advisory Committee to the Public Welfare Board. This committee will meet monthly at first, and never less than quarterly, with the Public Welfare Board staff and at least once

yearly with the Public Welfare Board itself. The duties of this committee will be to hear problems and grievances that arise from county welfare boards and the State Public Welfare Board regarding the practice of medicine and also to act as a liaison for the North Dakota State Medical Association and its members in problems that arise with welfare recipients and county welfare and state public welfare boards. This committee will be given an opportunity to iron out and to decide many issues of a medical nature that will arise during the administration of this program. It is felt that this committee has a great service to render to the profession and can be instrumental in improving our public relations with the Public Welfare Board, the governor, and the state legislature.

Negotiations for these various fee schedules were carried on by a negotiating team, consisting primarily of Drs. C. H. Peters, Bismarck, chairman; V. J. Fischer, Minot; James H. Mahoney, Devils Lake; Verl G. Borland, Fargo; E. J. Larson, Jamestown; O. V. Lindelow, Bismarck; M. A. K. Lommen, Bismarck; and R. P. Froeschle, Hazen. In addition, our president, Dr. John Fawcett, sat in on the last meeting with the Public Welfare Board and the governor.

One general meeting of the entire committee was held on December 12, 1959, at Fargo. The main purpose of this meeting was to report to the committee as a whole on the actions and work of the negotiating team and to ask for their approval. In addition, one resolution was passed by the Medical Economics Committee at its December 12 meeting in Fargo, asking the council to inform Blue Shield and Blue Cross that where grievances about the conduct of physicians in their relationship to prepaid medical plans could be documented, such grievances should be presented to the North Dakota State Grievance Committee for proper action. These problems arise in the field of use, abuse, and overutilization of Blue Shield, Blue Cross, and commercial insurance plans. If these organizations can document such instances and if they cannot be properly adjusted on a local level or with the Liaison Committee to Blue Shield and Blue Cross, then this material should be presented to the Grievance Committee for proper action. The Medical Economics Committee also recommended to Blue Shield and Blue Cross that a greater selection of deductible policies should be made available to the public. It is felt that deductible policies of \$50 and \$100 should be formulated, thus keeping premiums lower.

At the present time, there is no agreement with the Veterans Administration and no fee schedule exists between the North Dakota State Medical Association and the Veterans Administration regarding care and examination of their recipients. The relative value schedule is in effect with the Indian Agency of the Public Health Service and apparently is working very well and without any major problems arising. It apparently is satisfactory to both parties. Negotiations are actively proceeding with the North Dakota Division of Vocational Rehabilitation with the possibility that the relative value schedule will be adopted.

In addition to the foregoing items, continued effort has been made by the subcommittee, under the direction of Dr. Keith Foster of Dickinson, to continue our educational program of the public, the hospitals, and the physicians in North Dakota in the proper use of prepaid medical care plans. We believe that some headway has been made and that gradually there has been a clearer understanding of insurance principles and that the public and physicians in general are beginning to understand that proper use of these plans is essential to their preservation.

Abuse by a few physicians causes marked deterioration in our public relations for the entire profession and makes it extremely difficult to insure the proper climate at the time of negotiations for new fee schedules.

I again, as chairman of this committee, wish to thank all those previously mentioned who have participated actively in this program for their very close and intensive cooperation during this past year.

C. H. PETERS, M.D., Chairman

Committee on Rural Health

The number one problem facing the Rural Health Committee is getting and keeping physicians for rural communities. As in any problem, this one has many facets. However, it is our feeling that lack of hospital facilities poses the greatest problem. We do not feel that it would be feasible to build a large number of small hospitals and one-doctor hospitals throughout the state to alleviate this problem. However, by making use of the existing facilities, the building of nonfeasible units would be avoided. This could be done by having open-staff hospitals in the hospitals already existing in the state. As this report is being written, the problem has erupted into the lay press. The medical profession is getting extremely bad publicity at a time when it desperately needs friends to combat the march of government medicine.

This committee would also like to see study groups established that would make a medical survey of an area or a community requesting a physician in order to give an interested physician the medical profession's viewpoint of the community. These study groups would be so arranged that the survey would not be conducted with doctors in the immediate community but preferably by physicians somewhat removed from the area.

In order to encourage North Dakota medical students to return to their state to practice, the Rural Health Committee would like to suggest that the preceptorship that the sophomore medical students at the University of North Dakota are required to serve at the present time be expanded to include service with rural physicians if the student so desires. In addition, we would like to see contact kept with the third year students in order that they may be encouraged to come back and serve preceptorships at the end of their junior year of medical school as paid assistants to rural physicians. This system is similar to that which is in operation in Nebraska at the present time.

This committee would like to thank Dean Harwood and his staff for all the work they have done in encouraging students to return to practice in North Dakota and for the extensive surveys they have conducted in communities requesting physicians.

This committee also wishes to include in its report the report of Arnold O. Goplen, director, Division of Hospitals and Legal Council for the State Health Department. This is as follows:

December 30, 1959

To: State Health Council and Hospital Committee of the State Health Council

From: Arnold O. Goplen, director, Division of Hospitals and Legal Council

Subject: Discussion agenda pertaining to hospital program for the meeting to be held January 11 and 12, 1960, at the Rehabilitation Center, University of North Dakota.

CURRENT STATUS OF FEDERAL GRANT CONSTRUCTION PROGRAMS

Projects under construction

1. St. Joseph's Hospital, Minot—replacement addition and extensive modernization. Construction started July

27, 1959. Estimated total cost is \$2,250,000. (Federal participation in the cost is limited to portion of replacement addition.)

2. Lutheran Home for the Aged, Minot—64-bed nursing home addition, 95 per cent completed. Estimated total cost is \$615,000. Will open April 4, 1960.

3. Grand Forks Home for the Aged—50-bed nursing home addition, 95 per cent completed. Estimated total cost is \$536,000. Opened February 3, 1960.

4. Jamestown Crippled Children's Hospital School—rehabilitation project including dietary, homemaking, and prevocational units, 95 per cent completed. Estimated total cost is \$238,300. In operation.

Projects not finally audited but substantially completed and placed in operation

1. Trinity Hospital, Minot—43-bed chronic disease unit and extensive expansion and modernization of the general hospital. Substantially completed on October 21, 1959. Estimated total cost is \$2,500,000. Federal participation in cost is limited to chronic disease unit.

2. Kenmare Deaconess Hospital—replacement of 36-bed general hospital. Substantially completed and opened for patients on March 20, 1959. Estimated total cost is \$518,800.

3. St. Aloisius Hospital, Harvey—replacement of 55-bed general hospital. Substantially completed and opened for patients on December 14, 1959. Estimated total cost is \$1,032,000.

Projects which have received preliminary federal approval

1. Golden Valley County Hospital, Beach—replacement of 24-bed General Hospital. Construction start proposed early in spring of 1960. Estimated total cost is \$415,600.

2. Tioga Hospital and Clinic—new 26-bed general hospital. Construction start proposed early in spring of 1960. Estimated total cost is \$425,000. Bids opened April 29, 1960.

3. Garrison Memorial Hospital—addition of pediatric floor to serve Indian patients. Bids for this expansion were opened December 16, 1959, and the proposals are currently in the hands of the Indian Health Service for final approval. Funds for this project will come exclusively from the Indian Health Facility Construction Fund, Public Law 85-151. The estimated total cost of this project is \$244,000. Construction started April 1, 1960.

Projects scheduled for construction during 1960 based on recommendations of State Health Planning Committee on October 30, 1959, and approval of State Health Council, on November 7, 1959.

1. St. Luke's Hospital, Fargo—34-bed chronic disease unit and extensive expansion and modernization of general hospital. Anticipated construction to start spring of 1960. Estimated total cost is \$1,303,600. Federal participation in cost is limited to chronic disease unit and some replacement of beds for acute cases.

2. St. Alexius Hospital, Bismarck—24-bed psychiatric unit and expansion of administrative, surgical, and pediatric services. Construction anticipated to start the spring of 1960. Estimated total cost is \$1,798,490. Federal participation in cost is limited to psychiatric unit.

3. McIntosh County Memorial Hospital, Ashley—12-bed addition to general hospital. Construction anticipated to start the spring of 1960. Estimated cost is \$108,120.

1. Turtle Lake Community Hospital—12-bed addition to general hospital and modernization and expansion of service facilities. Construction anticipated to start the spring of 1960. Estimated total cost is \$180,000. Federal participation in cost is limited to the new addition.

5. Luther Memorial Home, Mayville—a 42-bed nursing home and a 20-bed boarding unit for the aged. Construction anticipated to start the spring of 1960. Estimated total cost is \$534,200. Federal participation in cost is limited to the nursing home portion.

6. Central Dakota Nursing Home, Jamestown—a 100-bed nursing home. Construction anticipated to start the spring of 1960. Estimated total cost is \$715,000.

CURRENT STATUS OF LOAN APPLICATIONS UNDER NORTH DAKOTA SENATE BILL 290

Nursing home projects approved by State Health Council, November 7, 1959

1. Stanley Retirement Home—approval of application for \$100,000 loan for construction of a 39-bed nursing home at Stanley. Contracts awarded will be based on bids opened September 17, 1959. Estimated total cost is \$328,000.

2. Baptist Home for the Aged, Bismarck—approval of application for \$100,000 loan for construction of 35- to 40-bed nursing home addition to Baptist Home for the Aged at Bismarck. Architectural drawings in process of development for construction to start the spring of 1960. Estimated total cost is \$423,000.

Pending applications filed with the State Health Department

1. The Harold S. Haalands' Home, Rugby—filed application December 14, 1959, with the State Department of Health for a \$100,000 loan to construct a 68-bed combination nursing home and home for the aged. Preliminary architectural plans have been approved, and the application was referred to the Bank of North Dakota on December 29, 1959. Construction is proposed during spring of 1960. Estimated total cost is \$500,000.

2. The Lutheran Sunset Home at Grafton—filed an amended application December 22, 1959, with the State Department of Health for a \$100,000 loan to construct a 40-bed nursing home. Preliminary architectural plans were filed with the Health Department on December 24, 1959. Conferences regarding plans are in progress.

3. Parkside Lutheran Home, Lisbon—filed application November 7, 1959, with the State Department of Health for a \$100,000 loan to construct a 60-bed combination nursing home and home for the aged at an estimated total cost of \$350,000. Architectural plans have not to date been presented to the department for review.

Other anticipated loan applications for nursing home construction not on file with the Health Department as of this date

Devils Lake, New Rockford, Strasburg, and Westhope.

PROSPECTIVE CONSTRUCTION DEVELOPMENTS

1. Demand is increasing for expansion and modernization of existing hospitals, especially with emphasis toward increasing the areas and facilities for services. Examples: Good Samaritan Hospital, Rugby; Trinity Hospital, Jamestown; St. John's Hospital, Fargo; and Deaconess Hospital, Grand Forks.

2. Replacement of outmoded facilities. Examples: Crosby, Langdon, and Mandan.

3. Additions to relatively new general hospital facilities to provide more space. Examples: Cavalier, and LaMoure.

4. Promotion of additional psychiatric units in general hospitals. Example: Trinity Hospital, Minot.

5. Request for construction of student nurse residence for St. Michael's Hospital, Grand Forks.

6. Possibility of request for construction of new general hospital at New Town. This is related to the Indian

problem and the use of Indian Health Facility Construction Funds.

7. Possibility of expanding rehabilitation facilities at the University of North Dakota and at the Jamestown Crippled Children's School. Federal Funds currently available include an estimated balance of approximately \$12,000 of 1959 fiscal year funds in addition to the 1960 fiscal year allotment of \$55,650.

M. S. JACOBSON, M.D., Chairman

REPORTS OF SPECIAL COMMITTEES

Committee on Mental Health

Your Committee on Mental Health did not meet formally this year, since the state president appointed a 3-man liaison committee to work with the State Board of Administration. It was felt best to allow these men to act without interference. They have managed to aid in getting some psychiatric personnel at our State Hospital.

Individuals of the state society have aided in forming mental health organizations in both Fargo and Grand Forks this past year.

Your committee chairman, Dr. C. H. Peters, and Dr. M. J. Johnson met with Dr. Robert Hewitt of the United States Public Health Service concerning the mental health survey which the United States Public Health Service is making for the Legislative Research Committee. Also, society members were contacted in their home town by this research group.

As a delegate to the Governor's Conference on Children and Youth, an attempt was made to present to our study group the fact that our mental health program on a state level had suffered a serious setback in the spring of 1959. This was fairly well suppressed in the publications that were forthcoming, but it was brought out in the discussion groups. Dr. L. G. Pray chaired a group on the emotional needs and was able to also bring out some of the problems that we have in our state.

In looking back on the past year, I feel that the mental health program in the state is better off than it was last year at this time but that a lot remains to be done. The following year will be a legislative year. Thus, this society will have to be on its toes to see that proper gains are made in the mental health program on a local and state level.

K. G. VANDERGON, M.D., Chairman

Committee on Cancer

The Committee on Cancer wishes to reiterate that cancer is reportable, similar to communicable diseases. Medical librarians in the various hospitals will be instructed to have available suitable blanks for reporting such cases. In spite of repeated comments, there have been no appreciable improvements in reporting cancer. We also would like to urge staff members, and especially surgeons, to extend to the medical librarians their fullest efforts and cooperation in supervising the cancer registry. Pathologists working in the hospitals or in adjacent clinics are doing excellent work in maintaining and supervising a cancer registry, whereas other less fortunate hospitals are not keeping their cancer records current. The time is not too far distant when a complete and accurate cancer registry will be a requirement for accreditation. As you all know, it is now a requirement of the American College of Surgeons.

In addition to several state cancer meetings during the past year, your chairman attended a national cancer meeting in New York and a district cancer meeting in Portland. Apparently, the treatment of cancer has now

reached its peak in surgery and radiation, and all are anxiously awaiting the time when a breakthrough in the cancer horizon will present itself. The sessions during 1959 were centered around the recognition and treatment of precancerous lesions. Much emphasis was placed on leukoplakias, senile keratosis, polyps of the bowel, carcinoma in situ, hyperplasia of the endometrium, persistent gastric symptoms, cytology of cervical smears, bronchial smears, and gastric washings. One is inclined to glean from the lectures that the first breakthrough would be in the fields of leukemias and lymphomas. There appears to be a definite connection between leukemias and viruses. Dr. M. M. Hargraves of the Mayo Clinic, a national authority on leukemias, feels that there is a strong possibility that leukemias are linked with the agriculture and cattle raising industry. Five midwestern states, which include North Dakota, have the greatest incidence of leukemia in the country.

The North Dakota division of the American Cancer Society has sponsored for the past year a project in cytology which has been supervised by the Grand Forks County Medical Society. A complete report on this project will be presented in this column at a later date. So far, the project has moved along smoothly and is being accepted by the laity and Doctors alike.

The American Cancer Society has voted a grant-in-aid to Drs. Cornatzer and Miroff of the Medical School at the University of North Dakota to continue their work with cancer in mice.

The Cancer Caravan will again roll this spring at the request of 5 cities. The topic chosen this year is "Leukemias and Lymphomas." It, no doubt, will be one of the best presented. Dr. M. M. Hargraves of the Department of Medicine at the Mayo Clinic will present a paper entitled "Environmental Factors in the Production of Leukemias and Lymphomas." Also accompanying the caravan will be Dr. Chris A. Pascuzzi of the Department of Pathology at the Mayo Clinic. Dr. Pascuzzi's paper will be entitled "Classification and Pathology of Lymphomatous Diseases." These speakers will be presented at Minot on April 4, Williston on April 5, Dickinson on April 6, Bismarck on April 7, and Jamestown on April 8. The North Dakota Cancer Society will continue the policy of presenting speakers to the various societies upon request, and, if the dates coincide, a continuation of the Caravan will be carried on indefinitely. Some of the medical societies are unable to fit the Caravan schedule in with their regular meetings. In these instances, they are allowed to obtain their own speakers at the expense of the State Cancer Society. This has proved very successful and enables most of the medical societies to hear at least one paper on cancer during the year. Also continuing the past policy of furnishing speakers for the state obstetric, surgical, and medical society meetings, the North Dakota Cancer Society will again furnish cancer speakers during the 1960 season.

Doctors are again requested to make their services available as speakers for the various county units of the state cancer organization. In the future, physicians will be contacted by local workers or some of us who are on the committee, requesting them to speak on cancer when it is convenient. Suitable handbooks will be furnished by the state cancer office which contain excellent topics and require very little in the way of preparation. We thank you in advance for any cooperation you may give your county units.

Among recent honors coming to North Dakota was the appointment of Dr. L. W. Larson to the Board of Directors of the American Cancer Society. Dr. Larson

has been an ardent worker in the field of cancer and, in fact, is one of the pioneers for the North Dakota Cancer Society and is well deserving of this honor.

CARROLL M. LUND, M.D., Chairman

Committee on Veterans Medical Service

The Veterans Medical Service Committee held no meetings during the past year, and no problems were presented to the committee for study. So far as is known, relationships between the Veterans Medical Service and the North Dakota State Medical Association are at an even keel.

A. C. FORTNEY, M.D., Chairman

Committee on Nursing Education

No formal meeting of the Committee on Nursing Education was held in the year 1959 because no business or activities demanded such action.

On April 19, 1960, a meeting of the North Dakota Careers Committee, formerly the North Dakota Nurses Enrollment Committee, was held at Grand Forks. Another such meeting is to be held in Bismarck on the third Tuesday of June.

C. R. MOXIZ, M.D., Chairman

Committee on Maternal and Child Welfare

The Committee on Maternal and Child Welfare met in Fargo on February 20, 1960, and submit the following for your consideration:

1. We recommend that public health nurses be trained and equipped to handle emergencies in the event antibiotic reactions occur when giving antibiotics under a doctor's orders but without his direct attendance. The nurses should have available the equipment and medications to effect immediate treatment in case of antibiotic reactions. The procedure that the public health nurse will follow will be specifically outlined by each private physician requesting this service.

2. We recommend that a study be made of laws pertaining to the report of vital statistics as related to obstetrics.

3. We recommend the adoption of a statewide perinatal mortality study by the Maternal and Child Welfare Committee of the North Dakota State Medical Association with the cooperation of the Maternal and Child Welfare Division of the State Health Department. When the state Association approves of the principle of the program, the Maternal and Child Welfare Committee will then assume responsibility of providing forms, data, and information based on study programs throughout the country in an effort to make our program coincide with the findings of those in other states. The actual program of reporting will be placed in effect upon the conclusion of the Maternal and Child Welfare Committee's preliminary study and when the physicians of the state have been appraised of the program and the necessary forms.

R. E. LUCY, M.D., Chairman

Committee on Diabetes

The Committee on Diabetes, initiated by Dr. Leonard Larson of Bismarck, has continued its primary function of encouraging and coordinating annual diabetes detection drives throughout the state under sponsorship of constituent local medical societies. This function is a cooperative effort in support of National Diabetes Week, sponsored in November of each year by the Committee on Detection and Education of the American Diabetes Association, Inc. A trend of increasing participation is in evidence among district medical societies throughout

the nation. Last year, well over 1,000 societies held individual detection drives.

In November, 1959, only 1 detection drive was held in North Dakota, namely, the Kotana District Medical Society under the direction of Dr. Alan K. Johnson, chairman of the Committee on Diabetes for this society. Publicity was released the early part of November through National Diabetes Week. Newspaper, Television, and radio media were utilized, and announcements were made at civic meetings and posters were distributed.

The area covered included Divide, McKenzie, and Williams counties. Both Clinistix and Denco Sugar Tests were used as the testing unit, 638 by the Denco Sugar Test and 2,069 by the Clinistix method for a total of 2,707. Among this group, 27 positive tests were reported. Two of these patients were previously known diabetics. Unfortunately, it is a common practice among persons with known cases of diabetes to submit tests in the diabetes detection drives.

There were 25 people with positive tests in whom diabetes was previously unknown who were referred to their physicians for follow-up study. Of this number, 16 are considered to have had adequate follow-up reports thus far from the physicians to whom they were referred. Among this group, 3 new cases of previously unknown diabetes mellitus were diagnosed. It can be expected that in all likelihood at least 1 or 2 more cases will be disclosed from follow-up studies on the 9 patients with positive tests who are yet to be examined by their physicians. Nevertheless, the results of the Kotana District Drive have thus far more than justified the effort put forth.

A detection drive was approved by the Grand Forks District Medical Society for November 1959, but, after the committee met and discovered that the American Diabetes Association is no longer distributing individual Clinistix testing units which facilitates easy testing and easy reading, the drive was postponed until next year. The main objection in having to carry out a drive under the present regulations was that, contrary to last year, all tests would have to have been performed by local testing stations. Last year, it will be recalled that Clinistix testing kits were distributed to the patient. Each patient dipped the Clinistix in the urine specimen and promptly mailed it back to the medical society, where a group of volunteers simply opened the envelopes and read the tests as positive or negative. This is, of course, infinitely more practical than having to process specimens in testing centers.

Some district societies have voiced the opinion that an intensive detection drive once every two years is more feasible than an annual detection drive. There is no doubt in the mind of the committee that diabetes detection drives should be a permanent institution in the United States. There is dramatic emphasis in the fact that, in a recent symposium in the New York Academy of Medical Science describing current research in diabetes, a much higher incidence of diabetes has been found to exist than was previously supposed. For example, the number of known cases of diabetes in the United States is now considered to be 1,500,000, while the number of undiscovered cases is estimated to be 1,400,000. It will be noted that this represents nearly 1,000,000 more diabetic persons in the United States than were previously thought to exist. Additionally, it has been pointed out that nearly 9,000,000 people now living in the United States either have diabetes or the disease will develop in them. In view of the marked therapeutic advances that are continuing in the field of

diabetes research, it is of utmost import to seek out these unknown cases of diabetes and place them under adequate treatment.

It has been mentioned before in these committee reports that the Metropolitan Life Insurance Company has stated that the rate of increase in the diabetic population will be 3 times as great as the percentage increase in the general population during the generation ending in 1985. It is hoped that more district societies throughout the state will become interested in conducting detection drives in the future.

I wish to take this opportunity to thank the following members on the committee on Diabetes for their co-operation and continued interest in diabetes detection throughout the state: Drs. A. K. Johnson, Williston; W. H. Wall, Walhpeton; Kenneth Amstutz, Minot; P. Roy Gregware, Bismarck; Donald Barnard, Fargo; K. G. Foster, Dickinson; and B. Hordinsky, Drake.

E. A. HAUNZ, M.D., Chairman

Committee on Crippled Children

A meeting of the Committee on Crippled Children was held in Fargo, December 12, 1959. Following is a résumé of this meeting.

The first item on the agenda concerned the eligibility of children for Crippled Children's Services. It was the consensus of the several members present that a more specific definition be made of those children definitely indigent by more careful screening at the county level. The program is specifically designed for those needing free care, and, in most cases, the individual family should be given the opportunity to arrange payment of the physician's fee with an understanding that they are accepting charity when they apply to the Crippled Children's Services.

It is regretted that the general public does not more thoroughly understand the limitations of their various insurance policies in order to ascertain whether or not complete services are furnished the policyholders. Many people arrange for treatment for their children with their family physician or a referred specialist only to find that they must turn to the Welfare Board for help.

It is the belief of the committee that the child should first be examined by his family physician, who, in turn, should discuss financial arrangements with the family. In most cases, he will be aware of their ability to make payment and can send such a recommendation along to the specialist to whom he may recommend the child for care. It is felt that in most instances when the patient is not personally known, a check can be made of the local credit bureau or bank. If each physician would make this extra effort, it would greatly facilitate the handling of each case, as neither the family physician nor the specialist would be as apt to recommend the patient for welfare care if it is known that the family not only could but would be more willing to arrange financial details out of their own resources. This manner of prechecking would prove of infinite value to the Crippled Children's Services, which usually has no particular way nor the time to check the financial background of the patient and must take the word of the reports sent in from the various county agencies. It is most desirable that the physicians realize that it is far preferable to keep financial arrangements on a personal family basis than to refer patients not in actual financial need to the Welfare Board.

It is hoped that present negotiations with the Public Welfare Board in connection with the adjustment of the fee schedule will prove to be satisfactory. There are

certain inequities to be adjusted, and it is hoped that a 15 per cent over-all increase will be made. It was noted that hospitalization is now the largest expense, with costs being nearly doubled.

Utilization of the National Foundation is recommended whenever possible for indigent care. However, the program of the Foundation is now greatly curtailed and it no longer pays physicians' fees—neither surgical nor medical.

Dr. Johnson next discussed the cardiac catheterization team now functioning in Bismarck under a regional program. Although this team has only been in operation a short time, it has proved to be extremely satisfactory, and North Dakota physicians should encourage their patients to have work done locally, as it is not feasible to send them out of state if the work can satisfactorily be done on a local level. It was recommended that the committee work in this direction. The charge for a work-up in Bismarck is \$75 as compared to \$200 to \$250 at the Mayo Clinic.

The next item of discussion was the case load of Crippled Children's Services. The program continues to show some expansion, with the greatest amount of treatment being given for congenital heart disease and the orthodontic treatment for malocclusion.

The committee also felt that there should be no relaxing of the Crippled Children's Services' present policy of referring patients to specialists within North Dakota when adequate treatment can be obtained from them and that out-of-state referrals should be continued only if such services do not appear to be adequate within the state.

The work of the physical therapist employed by the Crippled Children's Services was next discussed. She serves primarily to (1) check current records to determine if the patient is getting the care recommended by the physician and to motivate the patient to resume care if it has lapsed, (2) carry out the physical therapy recommended by the physician and instruct the parents of the child in home care, and (3) act as consulting physical therapist for the others in the state. She is most valuable as a local contact person for liaison between the Crippled Children's Services and the local personnel.

Dr. Johnson next introduced Mr. William Unti, executive director of the North Dakota Society for Crippled Children and Adults who gave the following report concerning Camp Grassick and its activities.

The first item on the agenda was a discussion in regard to the PWBIM letter, which indicates that children who are to be approved for physical therapy at Camp Grassick must have the approval of a board-qualified orthopedist. Following a lengthy discussion, it was agreed by the members of the Medical Advisory Committee that this policy should remain in force.

The second item on the agenda concerned the medical forms used to receive information in regard to applicants for Camp Grassick. A number of suggestions were made by the committee for improving the forms. It was agreed that a new form would be drawn up and circulated among the committee members for their consideration and comments and then returned to the executive director for final approval.

The third item on the agenda was in regard to the follow-up reports on the individual children attending the therapy session. The committee indicated that the reports were extremely helpful and contained much valuable information. However, it was suggested that a condensed summary sheet accompanying the reports would be beneficial as an easy reference, and then, if

additional information is needed, the reports could be read in detail. It was also suggested that, if possible, the reports be completed and sent out at an early date, because the referring physician is usually waiting for the final report from camp in order to proceed further with the rehabilitation plans for the child.

The fourth item on the agenda was in regard to stimulating the interest of physicians by sending them a letter in the spring announcing the camping date, purpose of the program, and so on. It was the unanimous opinion of the committee that this policy be continued and that repetition should encourage better and earlier referrals.

The fifth item on the agenda was a discussion about holding a committee meeting at Camp Grassick during the therapy session. Everyone agreed that this would be extremely valuable, since many of the members have never seen this program in operation. It was agreed that the executive director and the camp director would extend a written invitation to each member of the committee to attend a meeting at camp at a date to be set later. The purpose of the meeting would be to evaluate the entire program after seeing it function first hand.

The sixth item on the agenda was a report by Dr. Foster, who pointed out that a subcommittee that had been requested earlier by Mr. Unti as an advisory group to the Speech and Hearing Council, which is an affiliate of the Easter Seal Society, had been appointed and that the committee had held a preliminary meeting. Dr. Foster said that, because of inclement weather, many could not attend. However, a preliminary draft of relationship has been made, and a future committee meeting will be held. He also indicated that he felt this was a very important step in establishing good relationships.

The seventh item on the agenda was a presentation of a possible cooperative program between the Easter Seal Society and the North Dakota Heart Association in providing outpatient physical therapy service in outlying areas where therapy is not currently available, such as in the Dickinson and Williston areas. The committee indicated that such a program should be established on the basis of the 2 organizations underwriting the cost of a therapist and, if fees were charged, the fees should be paid directly to the therapist and not to the organization underwriting the program. No definite action was taken.

The eighth item on the agenda was a report by Mr. Unti in regard to other programs of the society, such as the recreational camp program for severely handicapped children, camping program for severely handicapped adults, and the day camping program. In addition, he reported on the oral rehabilitation program, which has been set up in Jamestown, with all referrals to the program being made to the society which acts as the coordinator between the dentist, the physician, and the hospital. Parents make their own financial arrangements with the dentist, physician, and hospital.

PAUL JOHNSON, M.D., Chairman

Committee on Aging and Rehabilitation

In June 1959, Dr. John Fawcett, president of the North Dakota State Medical Association, appointed members of the Committee on Aging and Rehabilitation. The Chairman of this committee, Dr. T. E. Pederson, executive secretary of the association, Mr. Lyle Limond, and delegate to the AMA, Dr. Willard Wright, attended a meeting of the Joint Council to Improve the Health Care of the Aged, which was held in Washington, D.C., on June 12 and 13. This meeting was called to enlist interest of the participating organizations and to assist the organization of affiliates in the states with the intent

of developing patterns for joint efforts and joint planning and also to help delineate the role of the affiliates in the preliminary state conferences to the White House Conference on Aging. In October, the AMA sponsored a planning conference on the aging problem, which was in Minneapolis. Several members of this committee and other doctors of the State Association attended.

The first formal meeting of this committee was held in Fargo in December, at which time the role of the Association's Committee on Aging and Rehabilitation was discussed. Further action was held in abeyance until after the appointment of the Governor's Committee on Aging. The second meeting of the Association's Committee on Aging and Rehabilitation was held in Fargo on March 18, 1960, and all members except 2 were present. Guests who attended were Dr. Rodgers Dickinson, Dr. O. W. Johnson of Rugby, and Mr. Don Eagles of Fargo. After general discussion on this problem of aging, it was felt that one of the large tasks of this committee will be to stimulate interest in the subject within the entire profession and, most particularly, to accelerate such interest and programing on the district medical society level. With this in mind, a resolution will be forwarded to the House of Delegates at the May meeting to consider instituting a Committee on Aging and Rehabilitation in each district society. A resolution will also be forwarded to the House of Delegates requesting that the North Dakota State Medical Association participate in the study of this problem with the Joint Council to Improve the Health Care of the Aged, including representatives from the North Dakota Hospital Association, the North Dakota Nursing Home Association, and the North Dakota State Dental Association.

Governor Davis has appointed 11 members of the North Dakota State Medical Association to serve on the Governor's Committee on Aging. Four members are from this committee.

The scope and importance of the problem of aging has been well recognized by the parent body. The AMA has been very active in this field since 1955. Emphasis on the importance of this problem on the state society level has been somewhat tardy in North Dakota. For this reason, we feel an urgency to educate ourselves and our communities in the recognition that medical and health problems in aging and the aged embrace special social, economic, psychologic, physiologic, and occupational considerations. It becomes imperative that members of the medical societies foster efforts to: (1) stimulate a realistic attitude toward aging by all people, (2) extend effective methods of financing health care for the aged, (3) expand skilled personnel training programs and improve medical and related facilities for older people, (4) promote health maintenance programs and wider use of restorative and rehabilitative services, (5) increase medical and social economic research in the problems of aging, and (6) offer leadership and cooperation in community programs for the senior citizens.

Your committee respectfully enlists the aid, support, and cooperation of all doctors in North Dakota to become familiar with this problem, so that they may educate and counsel in their communities in this very important area.

The recommendations from this committee to the House of Delegates are as follows:

1. It was moved by Dr. Christoferson and seconded by Dr. Amstutz that this committee recommend to the House of Delegates that each district medical society form a Committee on Aging and Rehabilitation.

2. It was moved by Dr. Haywood and seconded by Dr. P. L. Johnson that the House of Delegates approve

the formation of a Joint Council to Improve the Health Care of the Aged through the leadership of the North Dakota State Medical Association. This council would have 3 representatives from each of the following: North Dakota State Medical Association, North Dakota State Dental Association, North Dakota Hospital Association, and the North Dakota Nursing Home Association.

T. E. PEDERSON, M.D., Chairman

Committee on Foreign Trained Physicians

Foreign medical graduates continue to enter the United States in relatively large numbers, although definite figures are difficult to obtain. An approximate tabulation can perhaps be made by studying the figures released by the Educational Council for Foreign Graduates. In 1958, 1,005 examinations were given; in 1959, 4,346 were given; and it is estimated that during 1960, 7,200 graduates will apply to take this examination. The ECFCG grants 2 types of certificates: a permanent one to those who make a grade of 75 or better and a temporary one for those whose grade is between 70 and 75. Practically all state boards which use this examination as an aid in screening foreign graduates will allow only those with permanent certificates to take their examinations.

The number of foreign graduates who were licensed in the United States during 1957 was 1,525; in 1958, 1,768; and while figures for 1959 are not as yet available, they will no doubt show an increase over 1958. During 1958 and 1959, 2,324 foreign graduates were serving as interns in hospitals in the United States and 8,392 serving as residents. Some large city hospitals have as many as 50 to 90 foreign graduates serving on their house staffs. After July 1, 1960, there will be no foreign graduate serving as either intern or resident in our United States hospitals who has not been certified by passing the ECFCG examination or attained a permanent license in the state in which his hospital is located. The Council on Medical Education and Hospitals has approved this ruling as has the House of Delegates of the AMA.

Under the terms of the Educational Exchange Act of the State Department of the United States, it is very easy for a foreign medical graduate to obtain an exchange visitor visa to come to the United States to do advance work in medicine as an intern or resident. All that is necessary is for a United States hospital to inform the State Department that it has an approved internship or residency available which it is willing to assign to a foreign medical graduate. While the visa is for one year, it is renewable year after year after year. Fullbright travel funds are often available to foreign medical graduates.

The number of cities in the United States offering examinations in 1960 will be 33 and in foreign cities, 51.

It is interesting to note the countries from which foreign graduates come. The 1958 and 1959 figures are as follows: Far East, 3,109; Latin America, 1,737; Europe, 1,480; and Middle East, 1,331—a total of 7,657.

It will be noted that the greatest numbers of graduates come from the Far East and Latin America where medical schools are not on as high a rating as most of those in Europe.

An article on the problem of foreign interns appeared in *U. S. News and World Report* for November 30, 1959, in which the director of the Educational Council was freely quoted. The failure rate given to foreign medical graduates now employed in the United States is 20 to 28 per cent. On analysis, this percentage is based on those who make 70 or over in the examination rather than 75, which is the real passing grade. The

failure rate is actually closer to 50 per cent in the United States and 75 per cent when the tests are given abroad when 75 is used as the passing grade.

The following letter was forwarded to the State Department in Washington, D. C., on February 27:

Educational Exchange Act Department
Department of State
Washington, D. C.

Gentlemen: My name is C. J. Glaspel, M.D., and I am Secretary of the North Dakota State Board of Medical Examiners. In addition, I am chairman of the Committee of Foreign Graduates of the North Dakota State Medical Association and am responsible for a committee report on foreign graduates to be presented to the House of Delegates at their annual meeting in April.

It is apparent from the limited figures we have access to that foreign graduates continue to enter the United States each year in large numbers either as exchange visitors or by means of an immigrant visa.

Will you forward me the approximate figures as to how many foreign medical graduates entered the United States in 1959 on an exchange visitors visa and on an immigrant visa and from what countries they migrate? How many returned to their native land when their training was completed? Are exchange students morally obligated to return to their native land at the end of three years when their training is completed? If so, do they? Is their visitors visa renewable year after year? Will foreign medical graduates who take the E.C.F.G. examination in their native land and fail this examination still be permitted a visa to enter the United States?

Very truly yours,
C. J. GLASPEL, M.D.

Unfortunately, at the time of the preparation of this report, a reply from the Department of State had not been received; therefore, it cannot be incorporated in the report.

During the past year, 7 foreign graduates were permitted to write the North Dakota State Board examinations, and 4 of these passed and were given a temporary license. Previous to this year, 45 foreign graduates had written the North Dakota Boards and 30 were licensed. This failure rate is much lower than exists in most states.

The North Dakota Medical Board will continue to screen and evaluate foreign medical graduates very carefully, with the purpose of admitting those who are well qualified to practice efficient medicine and rejecting those who fall below our standards of education and training.

C. J. GLASPEL, M.D., Chairman

Committee on American Medical Education Foundation

Our state American Medical Education Foundation (AMEF) program has been relatively successful in recent years in receiving contributions from about one-third of the physicians in the state, amounting to an average gift of over \$50 each. This success would indicate that our previous efforts have been effective in selling the needs of medical education.

A new method of contributing has been proposed that could supplement the present program, making it more convenient for many doctors to donate. This program would not replace the present efforts but would utilize the services of the Blue Shield office in Fargo to deduct the contributions from its physician payments in any amount prescribed to by the contributor.

The advantage of this method would be twofold: first, the doctor would be able to contribute any amount, large or small, amortizing the gift over a number of lesser payments; and, secondly, the contribution would be made from funds never actually received and, therefore, not as readily missed by the contributor.

From a fund raising standpoint, it would be an excellent method because it would tend to increase the amounts contributed and also make it more convenient to the contributor. Details of promotion would have to be

resolved by the AMEF Committee. The following immediate answers are:

1. This process is completely voluntary so that, if there is complete state endorsement of the program, each physician still has the option.

2. The amount can vary from small to large, with only a slight restriction necessitated by the mechanics of the administration.

3. The tax benefits would remain the same as any single straight cash contribution.

4. The gift can be designated to a specific medical school, and the medical school would be notified of the designated gift.

This is an effort on the part of the Blue Shield to assist our state AMEF program. For the Blue Shield directors to do this, however, the idea must be accepted by the state medical society. This acceptance must come from the districts first and then from the state level. I would like very much to have your assistance in presenting the Blue Shield program in each district. Between now and April 30, every North Dakota district will have at least 1 and probably 2 district meetings where this plan can be presented for discussion. If it is felt that further information is needed or that there are some specific questions about this approach for voluntary support of medical education, please let me know and I will try to explain it more fully. Please let me have your comments.

W. E. G. LANCASTER, M.D., Chairman

Committee on School Health

There was no official meeting of the Committee on School Health held during the past year.

The Governor's Committee on Children and Youth is to be commended for its work. Our association was represented on this committee and its recommendations for school health and children and youth may be found in the committee's booklet on page 7, which covers emotional problems, and on page 17, which deals with physical development.

R. W. McLEAN, M.D., Chairman

Liaison Officers to the North Dakota State Bar Association

The writer was one of approximately 22 physicians of the Bismarck and Mandan areas who attended a joint meeting of the North Dakota, South Dakota, and Montana bar associations, which sponsored Mr. Melvin Belli of San Francisco. Mr. Belli, who is one of the leading exponents of more adequate awards in malpractice suits, presented an address and discussion of interest chiefly to lawyers, although of considerable interest to the medical profession. He continually stressed the "silent conspiracy" of the medical profession in their unwillingness to testify in malpractice suits. This writer has constantly strived for a closer liaison between members of the North Dakota Bar Association and the North Dakota State Medical Association in following out the precepts of the Professional Code, which was formulated several years back. It is hoped that by this closer working relationship that most, if not all, malpractice actions can be avoided or settled prior to any public action.

It is also noted that the AMA is about to sponsor a program designed to acquaint physicians with the legal aspects and responsibilities of medical practice. It is trusted that this program can be beneficial and will be accepted by the physicians.

P. L. JOHNSON, M.D., Representative

Liaison Committee to the North Dakota State Dental Association

During 1959, there was no formal activity within the dental society.

DAVID G. JAEHNING, M.D.

Report of Representative to the Medical Center Advisory Council

This council met on June 24, 1959, and on January 29, 1960. Your representative attended both meetings.

Admissions. The graduating class of June 1959 consisted of 33 second year students, all of whom were satisfactorily transferred to 16 different schools. There were 40 who started this class. Of these, 3 withdrew, 2 were dropped, 2 were allowed to repeat the following year, and 3 received postsophomore fellowships and will transfer next year.

Forty-four students were admitted in the fall of 1959. Forty-two of them were North Dakota residents, and 2 were Minnesota residents who had been attending the University for their premedical work. Of these, 1 has withdrawn to attend graduate school, and the other eliminated himself scholastically.

So far there are only 36 North Dakota applicants for the freshmen class of the fall of 1960 compared to 56 at this time last year. To date, 19 of these have been accepted, 2 rejected, 4 deferred, and 1 has been lost to another medical school. There are 10 left to interview, and 5 of these were rejected one year ago. There are many nonresident applicants. Because of the apparent fall off in qualified North Dakota residents applying, the Medical Center Advisory Council passed a motion on January 29, 1960, requesting the State Board of Higher Education to accept more out-of-state students if there are an inadequate number of qualified North Dakota residents applying.

Student loans. During the last year, \$64,200 was loaned from the Medical Center Loan Fund, which provides up to \$75,000 a year. To date, from this fund, a total of about \$180,000 has been loaned to medical students. Six students who borrowed \$2,500 a year in 1957 and 1958 are obligated to return to North Dakota. So far, 3 of them have decided on locations within the state.

There are numerous smaller loan funds from which \$10,860 has been loaned, an average loan being \$450. Funds from these are nearly exhausted, but repayments should start coming in soon.

Biochemistry service. The biochemistry laboratory continues to carry out a large number of tests for physicians and hospitals throughout the state. This work is limited to unusual procedures, which generally cannot be performed elsewhere, and fees are charged for these procedures to cover cost. It is the intent of the Medical Center to discontinue such services as rapidly as they become available in other laboratories throughout the state.

Rehabilitation Unit. The number of patients cared for continues to increase slowly. Only 77 persons were served in the first year of operation. This number increased to 115 in the second year. Patients have been treated from all parts of the state and from Montana and Minnesota. The largest percentage of referrals has been by physicians, with Vocational Rehabilitation, Workmen's Compensation Bureau, Welfare Board, and a few other organizations supplying most of the rest. There have been 5 self-referrals, all of whom have been referred back to their local doctors.

The staff is a little short in psychology and speech therapy. Students are being trained in various departments.

The staff definitely feels that one of the biggest obstacles is lack of housing in the unit for many of the patients. The administration of the Rehabilitation Center would like very much to add 1 or 2 stories for housing and dining and teaching facilities. Under consideration is the addition of a curative workshop area and the aforementioned dormitory housing.

As presently operated, all patients referred are seen by a well-qualified internist, who is on the staff of the Medical School. They are also seen in consultation by one of the 3 competent orthopedists practicing in Grand Forks. In addition, arrangements have been made for consultation with other qualified specialists in various fields. Personal discussion with men acting as consultants indicates to me that, in their opinion, excellent work is being done in physical therapy, occupational therapy, vocational rehabilitation, and speech therapy. They believe that the quality of work merits the confidence of physicians and organizations throughout the area in referring patients.

It is agreed that a well-qualified physician trained in rehabilitation work would be a very desirable addition to the staff. Such men are extremely hard to find and are usually kept in the large centers where they are trained. The staff of the Rehabilitation Center is well aware of the advisability of such a person if and when one can be obtained.

Budget. Expenditures of Medical Center funds are very close to the incoming funds from the mill levy. The cost of operating the Medical School; the Rehabilitation Center, which is not yet self-supporting; the Nursing School; the Blood Bank; and various other projects that have been undertaken, together with a general increase in cost of operation, will not allow expansion of services.

There is still \$600,000 impounded by order of the state legislature a few years ago when the construction of another tuberculosis hospital was under consideration. These funds could well be utilized in the Medical Center itself when and if a future state legislature releases them.

Psychiatric training program. At present, 1 doctor is in training under this program. There have been 2 other applicants whose status in relation to the program is not settled.

The bill passed by the legislature setting up this program allows for the training of psychiatrists and allied personnel. The latter is not defined. The Medical Center Advisory Council and the administration of the Medical Center do not feel that Medical Center funds are sufficiently adequate to embark on training programs for persons other than psychiatrists.

P. H. WOUTAT, M.D., Representative

Report of Representative to the State Health Planning Committee

This committee held 2 meetings this past year, 1 during the summer and 1 on October 30, 1959. Your representative was out of the state for the summer meeting and was unable to attend the October meeting because of weather conditions.

Discussion with various committee members and review of the minutes of the meetings indicate that the philosophy of the committee has remained the same. In general, it is considered that few, if any, new hospitals are needed within the state. Emphasis should be put on nursing home facilities and expansion and renovation of existing hospitals.

Hill-Burton and related funds to aid in construction were granted the following: (1) a 24-bed psychiatric unit with supporting services at the St. Alexius Hospital, Bismarck; (2) a 31-bed chronic disease unit and 38 replacement beds at the St. Luke's Hospital, Fargo; (3) a 12-bed general hospital addition at Ashley; (4) a 12-bed general hospital addition at Turtle Lake; (5) a 100-bed nursing home at Jamestown, known as the Central Dakota Nursing Home; and (6) a 42-bed nursing home at Mayville, known as the Luther Memorial Nursing Home.

P. H. WOUTAT, M.D., Representative

Liaison Committee to the State Board of Administration

Two meetings were held between the Liaison Committee, consisting of Drs. G. A. Dodds, Edwin Boerth, and myself, in Jamestown with the State Board of Administration. Very little of a constructive nature was accomplished, but our full cooperation in improving the situation at the State Hospital was offered.

I believe the staff is complete at the hospital now, and we must let time determine whether the situation is adequately covered. The appointments to the staff were made without consulting this committee.

Your committee has steadfastly maintained that a physician should be superintendent of the State Hospital. However, we also recognize that Mr. Henry Lahaug is an outstanding administrator and under any regime will be indispensable to the institution.

JOSEPH SORKNESS, M.D., Chairman

Committee on Cardiovascular Diseases

The meeting was convened by the chairman, Dr. R. M. Fawcett, on December 12, 1959, at the Gardner Hotel, Fargo. Committee members present were Drs. R. M. Fawcett, chairman; Dr. R. D. Story; and H. C. Walker, Jr. Others present were Dr. Walter Pretorius, State Health Department; Mr. George Michaelson, traveling secretary, North Dakota Heart Association; and Mr. Lyle A. Limond, executive secretary, North Dakota State Medical Association.

The chairman outlined the functions of the committee. He felt that it should serve as a liaison committee between the North Dakota State Medical Association and the North Dakota Heart Association. Inasmuch as this committee's report would be in the *Handbook* of the House of Delegates of the association, each of the delegates would be informed of the committee's actions and recommendations. Dr. Fawcett also felt that the committee should be enlarged by the addition of 2 general practitioners and 1 pediatrician, so that it would be more representative of the association as a whole.

The committee offered the following suggestions:

1. Encourage the North Dakota Heart Association to expand its professional education program by underwriting the cost of speakers, films, and so on for state and district medical society meetings.

2. The membership of this committee should be made up from the several categories in medicine. The following committee make-up is suggested: 2 internists, 2 general practitioners, 1 surgeon, 1 pediatrician, and the chairman.

Mr. George Michaelson discussed 4 plans in meeting the need for physical therapy in rehabilitation of stroke patients in areas of North Dakota which are not presently served by a physical therapist. The plans are as follows:

1. The North Dakota Heart Association to cooperate with the North Dakota Easter Seal Society and/or with other voluntary health agencies on underwriting the cost of a physical therapist in areas not now served.

2. The North Dakota Heart Association to underwrite the guaranteed income and expenses of a physical therapist who would concentrate on CVA cases. The physical therapist would charge fees for services given and would be attached to an existing facility, for example, a hospital or medical clinic. The Heart Association would pay only the difference between the guaranteed income and the fees for service income.

3. The Heart Association to cooperate with the Crippled Children Services Division of the North Dakota Public Welfare Board in underwriting the cost of a physical therapist. This plan is similar to plan 1, except that special differences may exist because of state and federal regulations.

4. The Heart Association to set up training seminars for the county public health nurses who, in turn, would instruct a family member of CVA victims in performing home physiotherapy. The public health nurses would do follow-ups on the recommendation of the doctors. This proposal is similar to the plan now in operation in Georgia.

These various plans are still being studied by the North Dakota Heart Association. No conclusions or recommendations were drawn at this time other than to advise the Heart Association of the importance of keeping the control of the patient in the hands of the family physician.

The proposed Secondary Rheumatic Fever Prophylaxis Program was presented by Dr. Walter Pretorius of the State Health Department. Considerable discussion followed the presentation. Two principal conclusions were reached:

1. There is a need for secondary rheumatic fever prophylaxis in some children who are not receiving the drug because of the cost burden of long-term drug therapy.

2. The proposed program leaves the decision of participation in the program and control of the patient squarely in the hands of the family physician.

The following statement of approval was prepared for presentation to the Council, which met in the evening of December 12, 1959:

"The Cardiovascular Committee of the North Dakota State Medical Association approved the principle of a secondary rheumatic fever prophylaxis program and gave its approval to the program as presented by Dr. Walter Pretorius of the North Dakota State Health Department.

"This committee recommends that the North Dakota State Medical Association give its approval to this program pending the acceptance of the program by the North Dakota State Pharmaceutical Association and the North Dakota Heart Association."

In accordance with the decision of the Council of the North Dakota State Medical Association to refer the subject of a Secondary Rheumatic Fever Prophylaxis Program back to the Cardiovascular Committee, Dr. Fawcett appeared before the Executive Committee of the North Dakota Heart Association on Sunday, December 13, 1959, and requested that it undertake a study of the needs for such a program in the state as well as an information program to acquaint the physicians of the state regarding secondary rheumatic fever prophylaxis.

ROBERT M. FAWCETT, M.D., Chairman

Committee on Liability Insurance

No meeting of this committee was held during 1959, since nothing was called to the attention of the chairman to be considered by the committee.

R. H. WALDSCHMIDT, M.D., Chairman

Liaison Committee to Blue Cross—Blue Shield

The report of the Liaison Committee to Blue Cross—Blue Shield for the year 1959 will be given orally by your chairman to the House of Delegates convening in Grand Forks.

O. A. SEDLAK, M.D., Chairman

Report of Delegate to the American Medical Association

Your delegate attended all sessions of the House of Delegates in 1959 and serves the Association in the following capacities: member of the Council on Medical Services and chairman of its Committee on Medical Facilities, member of Tripartite Liaison Committee with American Nursing Home Association and American Hospital Association, member of Committee on Rehabilitation, member of Special Study Committee on Medical Education, and member of Surgeon General's Committee to develop principles for area-wide community health facility planning.

Dr. Louis Orr of Orlando, Florida, took office as president in June 1959. He will be succeeded as president in June 1960 by Dr. E. Vincent Askey of Los Angeles, California, formerly speaker of the House of Delegates. Dr. Leonard Larson, of Bismarck, currently chairman of the Board of Trustees, will be nominated for the office of president-elect at the June meeting in Miami. These men have ably represented the Association and we expect they will continue to do so.

In June 1959 the House of Delegates received and adopted, with minor modifications, the comprehensive report of the Commission on Medical Care Plans, which was prepared under the direction of Dr. Leonard Larson, chairman. This report contained a great deal of information on various types of medical care plans and is a result of long and careful study. It is a most interesting document, which everyone could read with profit. In general, it points out that there are presently in operation a wide variety of methods of providing medical services or, to be more exact, of providing the payment for medical services. Among other important conclusions, the report pointed out that "free choice of physician" is an important factor in providing good medical care. At the December 1959 meeting, in order to clarify and strengthen its position on the issue of freedom of choice of physician, the House also adopted this additional statement: "Lest there be any misinterpretation, we state unequivocally that the American Medical Association firmly subscribes to freedom of choice of physician and free competition among physicians as being prerequisites to optimal medical care. The benefits of any system which provides medical care must be judged on the degree to which it allows of, or abridges, such freedom of choice and such competition."

The House again concerned itself with the relationship between medicine and osteopathy. In general, there seems to be a growing tendency to consider it advisable for doctors of medicine to consult with and to endeavor to help in the education of osteopaths and osteopathic students.

In the field of medical education, the House approved a program of preparation for general practice, which

proposes a new two-year internship program, and it outlined the content of this program. At the December meeting, the House passed a resolution which approved the creation of a Special Study Committee on Medical Education. This committee was charged with the task of developing a scholarship program and making a general over-all survey of medical education and, particularly, of the possibilities of expansion in the future. It is becoming well-recognized that the length and cost of medical education is very burdensome, and there is a tendency for well-qualified students to go into other fields. It will be the duty of this Special Study Committee to report on the situation and make appropriate recommendations. All physicians are urged to continue to support the American Medical Educational Foundation.

Your House of Delegates continues to be concerned with problems of the interprofessional relationships of physicians with themselves, with hospitals, and with other agencies. In December, a resolution was passed reaffirming the stand taken by the House in 1951, which stated in part that "A physician should not dispose of his service in a manner permitting a third party to derive a profit from sale of his services."

The AMA continues to carry on an extremely active program in its various field of interest. It is to be remembered that a very large portion of the AMA budget goes for strictly scientific matters which are directed by the various scientific councils, committees, and bureaus. In addition, an active and stepped-up program is being carried out in the field of social economics. During the past year, there has been a marked increase in activities concerned with the field of aging. Seven regional conferences on aging have been held, and a national conference is being planned. Various members of the Association, including Dr. Leonard Larson, are actively engaged in preparation for the 1961 White House Conference on Aging. There have been many other meetings with related groups also interested in this problem. Out of it all has come a rather definite program as to the best method of caring for the various categories of the aging population.

In the field of prepayment, the Association has developed an entirely new Prepayment Insurance Committee, which also has held numerous regional meetings and proposes to hold a national Conference on Prepayment in May 1960. In addition, relationship between the AMA and various organizations in the prepayment field have been considerably improved. Considerable impetus has been given to the development of prepayment plans by the action of the federal government in passing legislation permitting federal employees to be enrolled in prepayment plans with premium participation by both individual and government.

Remodeling of the headquarters office at 535 North Dearborn Street, Chicago, has been virtually completed and should greatly increase the efficiency of operations. It is hoped that many changes in office procedure and personnel will result in more efficient operation.

In the field of national legislation, your Association continues to oppose the inclusion of doctors in the Social Security program. It is to be noted that while the Association actively opposes such legislation as the Forand bill, it is, in fact, in favor of many legislative proposals and gives them active support and valuable information through the Washington office.

Again, your delegate would like to point out the absolute necessity of all doctors becoming vitally interested in social, economic, and political affairs as they relate to the health of the people. There are a bewildering multi-

plicity of groups interested in some aspect of health care, and, if their efforts are to be most productive, it is absolutely necessary for doctors of medicine to assume leadership and help in the direction of such efforts.

I would like again to express my appreciation to the House of Delegates of the North Dakota State Medical Association for the opportunity of serving as your delegate to the AMA.

WILLARD A. WRIGHT, M.D., Delegate

NEW BUSINESS

DR. LARSON: I appreciate this opportunity to appear before you. I was glad to hear the letter from Doctor Blasingame. I am sure that he sent it to you with his very best wishes. I bring to you greetings from the Board of Trustees and the officers of the American Medical Association. I hope that all of you will have an opportunity to visit the AMA headquarters in Chicago and see what the Association has in its way of facilities and staff and the caliber of the men serving us. It is a big institution with between 600 and 700 employees. We have almost completed a complete renovation of the building. A great number of changes have been made, which we think make the building more usable and more attractive. It cost more than \$2,500,000 to do this, but we felt it would be better to utilize this building than to attempt to build a new one. Some suggestions have been made that we move to Washington, D. C., but the Board has not felt it wise to recommend such a move at the present moment. However, we anticipate purchasing some property in the nation's Capitol in case we need it in the future.

The Chair next called on Dr. Lancaster, who spoke briefly and then introduced Mr. Jack Ryon of the American Medical Association.

DR. LANCASTER: Last year was our most excellent year for the American Medical Educational Foundation. After the program has been in force nine years, it seems that more than 157 members should contribute. We have considered a plan whereby a few more could be brought into the program. It utilizes the Blue Shield. We have with us today Mr. Jack Ryon, who is in close touch with the American Medical Educational Foundation.

MR. RYON: Thank you, Dr. Lancaster. This program, which we call the Blue Shield program, was originally proposed as an alternate to a direct gift by the State of Connecticut. The state found that their laws would not permit a direct gift. Their officers proposed this Blue Shield program as a method whereby the medical men could support medical education. A member would be requested to contribute to medical education through deductions from his Blue Shield claims. These deductions would be voluntary. This plan has several advantages. The amount would be taken out in small sums. It would be a convenient way for the Foundation to collect funds for medical education, and contributions would increase. This program would have to be endorsed by the medical association, but it would not commit any member of the association to contribute. It would increase the direct contribution to the school, and it could be given as a direct gift to the American Medical Educational Foundation.

This approach has a potential which would make contributions from the physicians truly significant to the support of medical education. We think it has a lot of possibility. What we need is an endorsement by the medical association. It would be no problem for the Blue Shield to administer it. It would be lots easier to

sell to the doctors. They would be asked for contributions in smaller amounts but more frequently.

The foregoing being an item of new business, the Chair referred it to Reference Committee No. 6 for consideration, of which Dr. DeCesare is chairman.

At this time Dr. Dodds introduced Mrs. John M. Van der Linde, president of the Woman's Auxiliary to the North Dakota State Medical Association.

MRS. VAN DER LINDE: Mr. Speaker, members of the House of Delegates and guests, I bring you cordial greetings from our members. Thank you for allowing me the privilege of presenting to you this summary of the accomplishments of the Woman's Auxiliary in the past year.

Our membership at the present time totals 320. Our districts number 10, as do yours, and, for the most part, our committees parallel yours.

At the national convention in Atlantic City in June of 1959, we received a Certificate of Meritorious Service from the American Medical Educational Foundation. North Dakota was one of 10 states so honored for high per capita contributions. The project for this year was the sale of stationery, and, with only 4 districts having reported to this date, the sum on hand is \$432.71.

The Sophomore Medical Student Loan Fund enjoys top priority as a fund-raising project. Since its inception in 1950, close to \$18,000 has been raised by district auxiliary projects and individual contributions.

At the convention in Atlantic City, North Dakota received an Honor Certificate from *Today's Health*, along with 19 other states, as a result of our increase in subscriptions. This increase was due, in part, to the 168 individual one-year subscriptions given by the North Dakota State Medical Association to our state legislators and, in part, to our own accelerated efforts.

The American Association of Physicians and Surgeons Essay Contest was conducted for the third consecutive year. This contest is gaining in popularity, and our ultimate aim is to reach every student in every high school in the state.

Our state publication, *News, Views and Cues*, has been completely revamped during the past year. Through the combined efforts of our managing editor, business and circulation manager, co-editor, and publicity chairman, this newsletter is sent at regular intervals to our entire membership. Included in it are articles prepared by various state chairmen, editorials, district news items, and alerts. We are delighted with our new format and proud of our official state publication.

Our Paramedical Careers program is most active in several districts. Two of our 5 clubs have recently received national charters from the National League for Nursing in New York.

This year, legislation was probably our most active program. At the request of the North Dakota State Medical Association, our members engaged in an active write-in campaign to present medicine's views on the Forand bill to our state and national legislators. Resolutions were drawn up in the districts and sent to the chairman and members of the House Ways and Means Committee in Washington, to our state legislators, and to our national representatives in both the Senate and the House. The suggested outline for action, which was given to each member, was followed faithfully in most districts. The response was immediate, wholehearted, and, we trust, effective.

Our revised Bylaws, patterned after the Bylaws of the National Auxiliary, will be distributed at this convention. This revision, undertaken last year, has just recently been completed in printed form.

Community service is a project in which we are all actively participating. Auxiliary members serve as volunteers and leaders in most endeavors relating to health, health education, and welfare. As a group, we make a tremendously large contribution in time and effort on local community activities.

For the first time, the auxiliary to the Student American Medical Association will be officially represented at this convention. We extend a most cordial welcome to them, and know that our closer association will be mutually beneficial.

We have looked to you for authority, guidance, and assistance. We are most appreciative of the cooperation we have enjoyed from you; from Mr. Limond, your executive secretary; and from his staff. We count it a privilege to work as your auxiliary, and we will continue to strive to serve you well.

Dr. L. G. Pray, delegate to the White House Conference on Children and Youth, next presented his report on a meeting of that conference.

DR. PRAY: This report will be quite brief. I did appreciate attending as a delegate the Conference on Children and Youth. There were excellent speakers, and every delegate had an opportunity to participate and express his own ideas. The history of these conferences goes back to 1909, when Theodore Roosevelt personally invited 200 leaders to meet with him at the White House to discuss with him the care of dependent children. That conference resulted in the Children's Bureau. Since that time, a conference has been held every ten years. This one was called about two years ago. The conference was preceded by state conferences and meetings of committees appointed by governors of every state. In North Dakota, Governor Davis appointed a committee of 110 people in various walks of life, and on this committee were 3 physicians, Dr. Tudor, Dr. Vandergon, and myself. This committee had a meeting in Bismarck last June, and it was subdivided into subcommittees to study the problems of youth, education, physical welfare, and juvenile delinquency. I was appointed to the Committee on Emotional Needs of Youth. Each committee chairman was to make a survey of the particular needs in his area and report back to the committee in October. At the conference in October all of the committee reports were discussed, and a complete report was sent to Washington to a planning committee. Late in the fall, members were appointed to the Conference in Washington, to consist of at least 27 members and not more than 40. This was an excellent group of people from all walks of life. It was a very worthwhile experience in Washington. The opening session was in the Maryland Field House. The highlight of the opening session was the appearance of President Eisenhower. He looked very fit and gave a very interesting talk. It was quite an impressive ceremony when he was ushered in. This was followed by meetings throughout the week. I will not attempt to go into the general scope of the conference. I have a paper to be published in *THE JOURNAL-LANCET*. The main purpose of this conference was to bring back instructions to the state for a permanent organization to count on in helping our youth in every way we can.

Dr. Glaspel, secretary of the Board of Medical Examiners presented a supplemental report of that board to the House of Delegates.

DR. GLASPEL: The Board of Medical Examiners is very appreciative of the opportunity you have given me to appear before you. We are happy that we can discuss some of the complex problems that we have in the field

of medical licensure with the House of Delegates and the medical profession in general.

Some states in this area that have medical practice acts do not permit anyone to practice medicine in their state except graduates of schools in the United States. If any of you have read my report on the foreign graduates problem, you will note a copy of a letter that I directed to the State Department in February relative to the student exchange in which I am interested. I waited for a reply, but the reply told me very little. This is the second time that I was given the run-around. Several years ago, I was attempting to write a paper regarding foreign medical graduates, and I did not receive any reply from the State Department at that time. I would like to give you a little data which I was able to acquire from other sources.

I was told that, in the past five years, 33,000 foreign graduates have entered the United States on the foreign exchange program, occupying positions of interns and residents. This averages over 6,000 foreign graduates per year. I wanted information on how many returned to their native land; for under the regulations of this program, the men are required to return to their native land at the end of five years of medical education. I believe not many return, but the State Department would not, or could not, give me these figures. Certain rules also specify that an exchange visitor, if he does return to his native land, cannot return to the United States for two years, but there are many loop holes in those rules. The attorney general has certain ways and means of waiving rules, thus allowing politicians to get onto various boards.

I cannot give you any figures as to how many return, but most of them stay in this country, since of course, living conditions are better for them here. In a way, this defeats the program, for, in the beginning, it was planned to have them return to their native land to elevate the health situation in their own countries. This failure to return has created ill will for nations which are already shy of well-trained medical men.

A unit has been developed by the American Medical State Boards, the American Hospital Association, and the American Association of Medical Colleges. This is a screening agency which has attempted to certify the records of foreign graduates. That is not an easy job. If their credentials are acceptable, they are asked to take a one day's examination, consisting of 360 questions. The mortality of these graduates has been about 20 per cent. Because the mortality rate was so high, the American Hospital Association was disturbed, and it was able to get a ruling that no foreign graduate could serve as an intern or resident in our hospitals who has not been certified as passing the examination of the Educational Council for Foreign Graduates, which require a grade of 70 or better, or who has not attained a permanent license in the state in which his hospital is located. The Council went on record asking the hospitals not to accept these men without this certificate. After July 1, any hospital accepting a foreign graduate who does not have a temporary certificate or a license will be immediately withdrawn from the approved list. We hope this will help a little, because there must be 8,000 to 10,000 foreign graduates who do not have this certificate.

The examinations are given in March and September. There are something like 7,200 applicants to take the examinations, which shows that they have found that they could not get a license. It is very easy for foreign graduates to enter this country. They can get a visa if they have an opening in an approved hospital, and they

can stay and get their visa renewed if they can find a hospital that will accept them.

I sometimes think that our Board is criticized because of our rather firm request for qualified and competent men. However, previous to this year, 45 foreign graduates have written the North Dakota Boards and 30 have been licensed. Our mortality rate is lower than in most states. We licensed 38 more last year and revoked no licenses. We have given temporary licenses to men who are not citizens. I think this temporary permit is a very good thing until they become citizens.

The next order of business was the announcement of the names of the Nominating Committee as given by the secretary, Dr. Naegeli, and are as follows: Drs. Hill, Naegeli, and Van der Linde. Dr. Hill is to serve as chairman.

Dr. Naegeli next presented 2 resolutions which the Council passed and wished to present to the House of Delegates:

RESOLUTION

Whereas, many physicians believe and various studies have shown that the goals, purposes, and accomplishments of the medical profession with regard to scientific, socioeconomic, and political developments in the field of medical care are poorly understood by the public, and

Whereas, greater public understanding and support are necessary if the best medical practice is to survive and continue to serve the public, and

Whereas, an effective public relations program is the best insurance that the purposes and performance of the medical profession will be understood and appreciated by the people of our state,

Now, therefore, be it Resolved that the North Dakota State Medical Association activate its program to expand and accelerate public relations through the North Dakota State Medical Association's Public Relations Committee.

RESOLUTION

Whereas, it is the time honored ethical practice of the physician to aid all who are in need of medical care, and

Whereas, the physician does not turn away the person in strained financial circumstances,

Now, therefore, be it resolved that the members of the North Dakota State Medical Association reiterate once again through the association's ruling body, The House of Delegates, that no person in North Dakota is ever refused medical care based on inability to pay.

These 2 resolutions were referred to the Committee on Resolutions, of which Dr. DeCesare is chairman.

Dr. Tudor next presented the following resolution to the House of Delegates:

RESOLUTION

Whereas, almost 500,000 persons are accidentally poisoned in the United States annually, and

Whereas, almost 1,000 children under the age of 5 years are accidentally poisoned daily by consuming household products containing harmful chemicals, and

Whereas, household products sometimes are not adequately marked so as to warn of their poisonous properties with the result that they sometimes are not stored out of the reach of children, and

Whereas, there is a great need for the education of the American people as to the dangers of accidental poisonings and the need to keep from the reach of children all household products which contain harmful chemicals,

Now, therefore, be it resolved by the Sixth District Medical Society that the North Dakota State Medical Association be requested to encourage the people of North Dakota to learn of the dangers of accidental poisoning and to take such preventive measures as are warranted by the seriousness of the danger, and

Be it further resolved that the present labeling law of the State of North Dakota be amended to include all types of household poisons.

Dr. Murray next presented the following resolution to the House of Delegates:

RESOLUTION

Whereas, during the past half century, scientific advances and improvement in community health services, as well as in the standard of living, have helped push back tuberculosis in the United States, and

Whereas, the United States Public Health Service and National Tuberculosis Association asked a small group of national leaders

in several phases of public health and tuberculosis control to define the major gaps in present practices and to suggest the action needed, and

Whereas, the major recommendation of this Arden House Conference on Tuberculosis, held at Harriman, New York, November 29 to December 2, 1959, was "The Ultimate Goal is Elimination of Tuberculosis in the United States of America," and

Whereas, the North Dakota Tuberculosis and Health Association feels that this goal can be reached by mobilizing all resources for a widespread application of the scientifically demonstrated and medically accepted procedures of adequate chemotherapy,

Now, therefore, be it resolved by the North Dakota State Medical Association that the recommendations of the Arden House Conference on Tuberculosis deserve the cooperation of every physician in North Dakota, and

Be it further resolved that public health authorities assume their responsibility for seeing that all tuberculous patients receive adequate treatment, and

that competent medical, nursing, and rehabilitation care of the tuberculous patient be the goal of every North Dakota physician, and

that the North Dakota State Medical Association actively support this program of tuberculosis elimination.

Speaker Dodds announced that the 2 foregoing resolutions would be given to the Committee on Resolutions for its action.

Dr. Nugent next spoke concerning his report of the Medical Advisory Committee to the Public Welfare Board. He felt that, since it contained some agreements which have been agreed to between the Public Welfare Board and the committee, he would like to have the report endorsed by the House of Delegates.

Report of the Medical Advisory Committee to the Public Welfare Board

As a result of the agreements negotiated by the Medical Economics Committee with the North Dakota Public Welfare Board, a Medical Advisory Committee was established to meet with representatives of the Welfare Board. A list of 16 physicians was submitted by the president of the association to the Welfare Board, from which the director of the board selected the following to serve on the committee: G. Christianson, Valley City; G. L. Countriman, Grafton; Joseph Craven, Williston; R. M. Fawcett, Devils Lake; R. W. Rodgers, Dickinson; V. J. Fischer, Minot; A. C. Fortney, Fargo; and M. E. Nugent, Bismarck.

An initial organizing meeting was held February 13, at which time a discussion of the responsibilities of the Medical Advisory Committee was held, and general plans, procedures, and organization were worked out. Additional meetings were held in March and April. The next meeting is scheduled in June, following which it is anticipated that meetings will be held on a quarterly basis, with the fall meeting coinciding with the meeting of the North Dakota Public Welfare Board.

At these meetings, the following points were agreed to between the North Dakota Welfare Board representatives and the Advisory Committee:

1. Points of difference between any physician and the welfare agencies could be communicated by a physician to any of the members of the Advisory Committee or the executive secretary of the state medical association for discussion at these meetings. Similarly, any differences which any of the welfare agencies wished discussed would be transmitted to Mr. Ralph Atkins to bring to the meeting.

2. Any changes in the medical policy of the North Dakota Public Welfare Board would first be brought to the Advisory Committee for discussion. Distribution of such changes will be through the office of the executive secretary to the members of the association.

3. On the medical health fee schedules, code (010) general physical examination was defined, and the title

of code (010) was changed to read "complete physical examination." A complete physical examination is to include a complete examination of the patient by a complete checking of all the systems. Any examination which is less than a complete physical examination is to be considered an office call, code (011).

4. Following surgery, the usual postoperative care shall continue for thirty days. This thirty-day period does not include medical complications. Each person hospitalized for more than thirty days will be considered a chronic case, and medical fees will be set accordingly unless the attending physician stipulates to the County Welfare Board to the contrary.

5. The committee recommended that the State Hospital at Jamestown should be used for psychiatric evaluations and treatment for inpatients for an extended stay. Outpatients should be treated at the closest available facilities.

6. The committee is to refer a purported problem in the area of "markup" by some ophthalmologists when glasses are provided through their offices rather than direct to the recipient from the optical company to the North Dakota Academy of Ophthalmology and Otolaryngology.

7. The committee agreed that all physicians are to be urged to use the facilities that are available in North Dakota before referring recipients out-of-state.

8. Considerable discussion was held concerning methods of reducing drug bills. It was recommended by the committee that the County Welfare Boards send a copy of the drug bill to the physician in cases in which the bill is greater than \$30. per month per patient. It was felt that in bringing the cost of the medications to the attention of the physician in charge, he might be able to devise ways of reducing drug costs.

9. A new, simplified P.W. 444 (medical report) was accepted. This is to be the only medical report form for the Welfare Board and may serve any of several purposes, which will be indicated on the form. The physician may not need to complete the entire form but will be required to furnish only that information requested on the face sheet of the form.

10. A revised P.W. 114 (medical authorization) was accepted. On this form, the sections dealing with diagnosis, prognosis, and treatment have been deleted.

11. It was recommended that the Medical Economics Committee work out a fee for services of a physician anesthesiologist when one is required for welfare recipients.

12. It was recommended that there be no allowance for assistant surgeon's fees.

13. To prevent the arbitrary deletion of certain items from the physician's charges, it was agreed to request the County Welfare Boards to return the complete form (PW 111) to the physician with an explanation as to why certain items are to be deleted before payment is made.

14. It was decided that when 2 or more physicians who are not joined in the practice of medicine are involved in caring for recipients requiring specialized services, separate authorizations are to be issued and these physicians are to be paid separately.

15. The Medical Advisory Committee is to write to any physician whose medical practices and/or charges in relation to welfare recipients are not in keeping with the accepted medical practice in his area. The physician is to be informed of this irregularity and asked to conform to the accepted practices of the other physicians in the state. The Board of Censors (Grievance Committee)

of a district medical society is to be used when continued medical problems involving a physician are reported by the County Welfare Boards.

The committee, after a review of evidence, has thus far written to one physician in the state, asking his cooperation.

M. E. NUGENT, M.D., Chairman

The Chair referred this report to the Committee No. 5, of which Dr. Mahoney is chairman.

Next followed a discussion concerning a Secondary Rheumatic Fever Prophylaxis Program as presented by Dr. Pray.

DR. PRAY: Dr. Walter Pretorius of the North Dakota State Health Department has been recommending to the doctors of the state that we adopt a Secondary Rheumatic Fever Prophylaxis Program. He took this up with the Council today, and the Council referred this to the House of Delegates for action. This program is recommended by the State Health Department and offers 3 primary services: (1) low cost medication, (2) regular check-ups by public health nurses, and (3) periodic reminders to the patients to return for care. This is purely a voluntary thing. Each case has to be referred by the patient's own physician. It is simply a matter of getting the patient with rheumatic fever to follow-up on his prophylaxis treatment. I can see no objection to this. It has been approved by the North Dakota Heart Association. I would recommend that the House of Delegates vote in favor of such a program.

Secondary Rheumatic Fever Prophylaxis Program

Purpose. The program is designed to prevent the complications of group A beta hemolytic streptococcal infections in individuals who have or have had rheumatic fever or rheumatic heart disease by: (1) providing additional tools to the physicians for maintaining prophylaxis, (2) providing low cost medication to patients on prophylaxis when needed, and (3) expanding lay and professional educational activities.

Authority. The administrative authority will reside in the North Dakota Health Department, either in the executive head or his duly appointed representative.

The legislative authority will reside in a committee composed of representatives of: (1) the North Dakota State Medical Association, (2) the North Dakota State Health Association, (3) the North Dakota State Pharmaceutical Association, and (4) the North Dakota State Department of Health.

The committee can be convened by direction of the executive authority of any of the foregoing agencies and shall be represented by the executive authority of each agency or a duly appointed representative. The chair of the meeting shall reside in the convening agency. Legislative changes shall require a majority agreement.

Violation of the rules and regulations of the program shall be corrected by the administrative agency. However, any decision or violation which any member agency feels deserves review may be reviewed by a simple written request by the executive authority of that agency directed to the administrative agency. The administrative agency will convene within thirty days a committee representing each participating agency.

The case will be reviewed by this committee and its decision will be final. A majority vote will be required to establish a violation by the defendant. A tie vote shall indicate nonviolation. Appropriate judicial action will be outlined by the committee.

Initial program. The program presented will be considered in effect when the 4 participating agencies'

executive authorities have provided written acceptance to the administrative agency. The participating agencies are: (1) the North Dakota State Medical Association, (2) the North Dakota Pharmaceutical Association, (3) the North Dakota Heart Association, and (4) the North Dakota State Department of Health.

Responsibilities.

North Dakota State Medical Association:

1. Case finding shall be the primary responsibility of the patient's physician. He will decide which cases are in need of prophylaxis. If a question of misdiagnosis arises, the case may be called to the attention of the North Dakota State Medical Association by any participating agency. However, adjudication shall be the sole responsibility of the North Dakota State Medical Association. A case must be withdrawn from the program at its request.

2. Eligibility for the program shall be determined by the patient's physician. Such cases must be accepted in the order of request by the administrative agency until the supply of medication is exhausted.

3. Low cost medication in the form of sulfadiazine (500 mg.) tablets, oral penicillin G tablets (200,000 or 250,000 units), and benzathine penicillin G (1.2 million units) IM will be available from participating pharmacists to eligible patients. Such medication will be dispensed by said pharmacists on receipt of a duplicate prescription marked "rheumatic fever program." Such prescriptions must not exceed a three-month's supply of medication and may not be refilled. In addition, the physician must submit to the administrative agency a form provided by the administrative agency, which will contain the necessary information to maintain an adequate rheumatic fever registry on participating cases. This form must be completed and submitted on the initial admission of the patient to the program and then, every two years hence, follow-up information must be provided for as long as the patient remains on the program. Any case not so registered with the administrative agency will not be eligible for low cost medication. The administrative agency will notify the participating pharmacists and physicians of cases improperly registered, and, after such notification, no further medication will be supplied until the case is properly registered.

4. Every case registered will be eligible for follow-up service. For patients not on medication, appointment cards will be made available by the health department, or the physician may use his own appointment card. When the patient returns, his appointment card or a duplicate will be placed in a provided health department envelope and mailed. A tickler file will be maintained in the health department. Whenever one of the appointment cards is overdue, the patient's doctor will be notified by the public health nurse and, if necessary, a follow-up visit will be made.

5. Follow-up services will be provided for all cases on medication with a lapse in prophylaxis. The nature of these services are defined later.

6. Routine public health nurse follow-up and home service visits will be available at the request of the physician. Those cases will be visited no less than every three months with a report of the visit to the patient's physician. These services are defined later.

7. Any registered case may be withdrawn from the program by a written request from the physician to the administrative agency.

North Dakota Pharmaceutical Association:

1. A participating pharmacist will be so designated on receipt by the administrative agency of his willingness

to participate in the program. These pledges will be available from the administrative agency and will be distributed by the North Dakota Pharmaceutical Association in cooperation with the administrative agency.

2. The pharmacists will dispense medication to eligible patients at a cost of not more than \$1.00 for a month's supply on the receipt of a properly filled out duplicate prescription.

3. The pharmacist will submit at the end of each month the copy of all duplicate prescriptions to the administrative agency in envelopes provided. At the end of each three-month period, the medication will be replaced by the administrative agency. If the pharmacists so request, medication will be replaced more frequently.

North Dakota State Department of Health:

1. Will be responsible for all administrative activities not otherwise designated to other participating agencies.

2. Will operate and maintain a current tickler file on all participating cases. The rheumatic fever central file or registry will be maintained by the administrative agency similar to the current tuberculosis registry in locked files. No information will be given to any agency or individual other than those responsible for maintaining the registry.

3. Will supply sulfadiazine, oral penicillin G, and benzathine penicillin G IM every three months to pharmacists only in order to replenish drugs used for cases until the supply of drugs is exhausted.

4. Will follow up any registered case which fails to maintain prophylaxis according to the records. Such a case will be deemed delinquent when a renewal prescription for medication is not received within thirty days of the expiration of the previous prescription. Follow-up will be by a letter to the physician first and, subsequently, to the patient, if necessary; and/or a personal visit to the physician first by a public health nurse or another designated official of the health department and, subsequently, to the patient, if necessary.

5. Will submit to the participating agencies not less than every two years an evaluation of the operation of the program and suggestions for any changes in rules and regulations.

6. Will provide educational services to lay and professional groups.

7. Will provide public health nurse follow-up services, where available, through district and county health agencies. Regular routine follow-ups on patients on physician's request will include: (a) follow-up visits on cases at least every three months, (b) physician report on each visit, (c) education of the family as to the importance of prophylaxis, (d) review with patient of faithfulness in maintaining prophylaxis, (e) assistance to family in procuring and using medication as prescribed, (f) notification of the physician and State Health Department of any irregularities or home or personal problems complicating prophylaxis maintenance, and (g) general health appraisal of the case.

Follow-up services will be provided within seven days of the request on delinquent cases, with subsequent reports to the physician and administrative agency.

Where necessary, and at the request of the local physicians, monthly clinics will be set up for the administration of IM benzathine G penicillin. These clinics must be attended by the physicians. No IM benzathine G penicillin will be administered by public health nurses unless a physician is in attendance.

Public health nurses will also refer to physicians any suspected case of rheumatic fever or rheumatic heart

disease discovered in school health or other case-finding programs.

Will provide forms, letters and so forth necessary for the operation of the program.

North Dakota Heart Association:

1. Will inform the physicians, pharmacists, and patients of the rheumatic fever prophylaxis program.

2. Will provide lay and professional educational services on the value of secondary rheumatic fever prophylaxis.

3. Will serve on the committees as designated for operation of the program.

The Chair referred this recommendation to Committee No. 4, of which Dr. Baumgartner is chairman.

Dr. Nugent presented another resolution to the House as follows:

RESOLUTION

Whereas, the importance of public education so as to combat the passage of additional social legislation is well-recognized by each physician, and

Whereas, the Council has recommended that a public relations program be developed by the North Dakota State Medical Association,

Now, therefore be it resolved that there shall be an increase in the annual dues to the North Dakota State Medical Association in the amount of \$25 per member, and

Be it further resolved that the money so obtained shall be used exclusively for the purposes of public information, public relations, and legislation.

This was referred to the Committee on Resolutions, as the matter of dues was on the agenda for the following day. At that time, it would be considered as such and recommendations from the Committee on Resolutions would be asked for at that time.

Dr. Murray next presented a final resolution to the House as follows:

RESOLUTION

Whereas, the North Dakota State Medical Association has never opposed the care of service-connected illnesses in Veterans Administration hospitals, and

Whereas, the North Dakota State Medical Association has not opposed the treatment of veterans in Veterans Administration hospitals if these people were indigent, and

Whereas, in recent years more and more nonservice-connected illnesses have been treated in Veterans Administration hospitals, and

Whereas, the great majority of these patients are responsible citizens with steady employment and should assume the same responsibilities for their medical care as do nonveterans,

Now, therefore be it resolved that the North Dakota State Medical Association join with over 25 other state societies in opposing the continued admission of patients with nonservice-connected illnesses to Veterans Administration hospitals, and

Be it further resolved that there be congressional investigation of this unnecessary spending of tax money, and

Be it further resolved that a copy of this resolution be sent to the American Medical Association and to the executive secretary of the State Medical Association of Maryland.

This resolution was also referred to the Committee on Resolutions.

Adjournment

There being no further new business to come before the House, it was moved and seconded that the first session of the House of Delegates adjourn to reconvene at 1:30 p.m. on Sunday, May 1, 1960. Time of adjournment was 6:00 p.m.

PROCEEDINGS of the House of Delegates

Of The North Dakota State Medical Association Seventy-Third Annual Meeting, Second Session

The second session of the House of Delegates was called to order by Speaker Dodds at 1:30 p.m., May 1, 1960, at the Dacotah Hotel, Grand Forks. The co-chairman of the Credentials Committee, Dr. Macaulay, re-

ported that a quorum was present. Secretary Naegeli called the role and the following delegates responded:

Drs.: L. G. Pray, Fargo; F. A. DeCesare, Fargo; E. J. Beithon, Wahpeton; W. L. Macaulay, Fargo; J. B. Murray, alternate, Fargo; D. W. Palmer, Cando; J. Mahoney, Devils Lake; F. A. Hill, Grand Forks; W. C. Dailey, Grand Forks; R. E. Mahowald, Grand Forks; R. C. Painter, Grand Forks; W. W. Frey, Drayton; F. D. Naegeli, Minot; A. F. Hammargren, Harvey; B. Iordinsky, Minot; M. W. Garrison, Minot; C. J. Klein, alternate, Valley City; M. A. K. Lommen, Bismarck; M. Nugent, Bismarck; R. B. Tudor, Bismarck; C. Baumgartner, Bismarck; E. Vinje, Hazen; J. N. Elsworth, Jamestown; J. M. Van der Linde, Jamestown; D. R. Strinden, Williston; R. E. Hankins, Mott; W. Hanewald, alternate, Richardson; R. W. McLean, Hillsboro; and J. Little, alternate, Mayville.

Twenty-five delegates and 4 alternates attended the session. Also attending the meeting were:

Drs. E. J. Larson, A. R. Gilsdorf, J. C. Fawcett, O. A. Sedlak, C. M. Lund, R. H. Waldschmidt, E. H. Boerth, R. W. Rodgers, C. H. Peters, T. E. Pederson, W. E. G. Lancaster, O. W. Johnson, G. Christianson, K. G. Vandergon, G. W. Toomey, V. G. Borland, R. D. Nierling, and Mr. Lytle Limond.

There being no objection, the reading of the minutes of the first session of the House of Delegates was dispensed with. Speaker Dodds then introduced Dr. Lund, president-elect, who, in turn, presented and introduced to the delegates, Dr. J. A. Myers, chairman of the Editorial Board of THE JOURNAL-LANCET, and Mr. Allan Stone, managing editor of THE JOURNAL-LANCET. Dr. Myers spoke as follows:

DR. J. A. MYERS: "We have just issued the ninetieth anniversary issue of THE JOURNAL-LANCET. That is quite a long time for a journal to exist in continuous publication. We are heading up now for the one-hundredth anniversary issue. I am so pleased to meet with you, and we want your advice as to what we can do to keep improving this journal from year to year. I am sure that you are all aware of what we have been trying to do. We are running as many scientific articles as possible. We want more of them. Your president has asked that every paper presented at this Annual meeting be presented for publication. There is nothing better in the country than your program, and we have always regretted the fact that we did not get more papers. Since these papers are prepared especially for you doctors in this state, we think they deserve publication. We hope this year to receive for publication every paper that is given here at the scientific program. We would also like to have you in the profession in North Dakota send us anything worthy of publication. The physician who submits a paper does not have to be in a larger city in North Dakota; sometimes a doctor practicing in a smaller community has interesting case histories.

We are developing "Profiles" of physicians who are 60 years of age or more. We are not getting enough of them from North Dakota. We do wish that some physician who has made some interesting contribution to medicine in your state would send in such an article with a photograph of the doctor, so that others may know and read about him. Mr. Allan Stone, who is managing editor of THE JOURNAL-LANCET, is here also to discuss the publication.

We would like to have as many of you doctors as possible attend our board meetings or give us any suggestions you have have to help us improve the publication."

Mr. Stone then addressed the Delegate.

MR. ALLAN STONE: "I consider it a real privilege to have this opportunity to speak to you about the work of THE JOURNAL-LANCET. We treasure this relationship that we have had over many years, and we trust that it will continue for many years to come. It is not often that one is associated with a journal that is celebrating

its ninetieth year, and yet it is a lusty, growing child. The journal is looking forward to growing all the time.

You will be interested to know that I have made a brief tabulation since I came up here and find that since January 1959, the journal has published 20 papers about members of your group. We have 6 or 7 additional papers from North Dakota ready for publication. This fits in very well with what we are attempting to present. THE JOURNAL-LANCET ranks at a high level with other journals throughout the country. We trust that we will have your continued support in the years to come. Perhaps we will all be privileged to stand here ten years hence and celebrate the one-hundredth anniversary of THE JOURNAL-LANCET.

It is not often also that a publication comes out on the day announced. We take great pride that the journal comes out promptly. I brought along a few copies of the May 1960, ninetieth anniversary issue, which is twice the size of the regular issue. We have an outstanding group of papers, 2 of which were written by members of your association in North Dakota. We hope that you will look forward to the May issue with a great deal of interest.

We are deeply appreciative of your support and we do hope that you will give us the benefit of your advice, counsel, and criticism. We also appreciate this opportunity to be with you and meet jointly with you."

Speaker Dodds expressed his appreciation to Dr. Myers and Mr. Stone for appearing before the House of Delegates. He noted that the contract for THE JOURNAL-LANCET was due for renewal but that this would be taken care of at the time the Reference Committee considering the report of the Committee on Official Publication made its report to the House.

The first order of business was the report of the Reference Committee to consider the reports of the president, secretary, executive secretary, and treasurer.

Dr. Naegeli, chairman, presented the following reports and their discussions, which were adopted section by section and as a whole:

1. *Report of the president.* The Reference Committee heartily joined the president in his commendation of the Committees on Medical Economics and Legislation and their chairmen, Drs. Peters and O. W. Johnson, who gave freely of their time and thought for the benefit of every member of the association.

The committee concurred in the desirability of improving the "public relations" of the association and each of its members by whatever means the appropriate committee thinks best.

The committee joined the president in reemphasizing the need for continued effort to defeat all legislation of the Forand type and to seek a mutually satisfactory solution to the problem of providing medical care for all segments of our population.

The committee agreed with the president that, in 1960 and 1961, more than ever before, it is urgent, yes—mandatory, that the Legislative Committee and every member of the association be alert to the economic significance of the political maneuverings both in Washington and in Bismarck and that all concerned be active on behalf of nonsocialized medicine. Once lost, the battle will not be fought again.

In regard to the distribution of the *Handbook* to the entire membership of the association, the committee wished to suggest that the Council consider the merit of this proposal after weighing its cost against the benefits, if any, to be gained.

The committee had no positive thoughts regarding the format of THE JOURNAL-LANCET but urged the membership of the North Dakota State Medical Association to contribute more papers of good quality to the journal, so that it may truly become the Medical Journal of North Dakota.

The committee concurred in the president's suggestion that the actions of our House of Delegates and Council be fully discussed in each district at the first meeting following the annual session of the association.

The committee heartily agreed with the president's suggestion that the first and second vice-presidents and the president-elect be kept active in committee work in preparation for their "presidential year."

The committee expressed its appreciation for Dr. Fawcett's untiring efforts on behalf of the entire membership of the association. This portion of the report was adopted.

2. *Report of secretary.* The Reference Committee reviewed the secretary's report and noted that the association acquired 3 times the number of new members in 1959 that it gained in 1958. It also noted that the perennial problem of late payment, or nonpayment of dues, continues to plague the secretaries in several of the districts. The Committee commended Dr. Naegeli for his work as secretary. This portion of the report was adopted.

3. *Report of the executive secretary.* The committee reviewed the report of the executive secretary and commended Mr. Limond for his labors in behalf of the association. His suggestions regarding legislative activity, public relations, medical care of the aged, and the seventy-fifth anniversary session of the association have already been commented upon by this committee or will be discussed by other committees. This portion of the report was adopted.

4. *Report of the treasurer.* The committee studied the several reports and statements of the treasurer and noted his careful stewardship of the association's funds. The committee commended Dr. Nierling for his diligence in the preservation and investment of these monies. This portion of the report was adopted.

The motion was made by Dr. Naegeli and seconded by Dr. Painter that the report be adopted as a whole.

F. D. NAEGELI, M.D., Chairman

M. A. K. LOMMEN, M.D., Vice-Chairman

R. C. PAINTER, M.D.

ELMER BEITHON, M.D.

Reference Committee to Consider the Reports of the Council, Councillors and Special Committees

Dr. Robert Tudor, chairman, presented the following reports and their discussions, which were adopted section by section and as a whole.

1. *Report of the chairman of the Council.* The Reference Committee reviewed the report of the chairman of the Council and approved the report. The Council should again be commended for its work during the past year. This portion of the report was adopted.

2. *Reports of the councillors.* The reports of the councillors were reviewed and approved. The councillors were commended for their fine reports. This portion of the report was adopted.

3. *Report of the Committee on Aging and Rehabilitation.* The report was reviewed and approved by the Reference Committee. This portion of the report was adopted.

4. *Report of the Committee on American Medical Education Foundation.* This report was reviewed and

approved. This portion of the report was adopted.

5. *Report of the Committee on Cancer.* This report was reviewed and approved. This portion of the report was adopted.

6. *Report of the Committee on Cardiovascular Diseases.* This report was reviewed and approved. This portion of the report was adopted.

7. *Report of the Committee on Crippled Children.* This report was reviewed and approved, with the reservation that the committee did not feel that it is practical or advisable for the family physician to screen patients to determine whether they are indigent.

A discussion followed this report concerning the advisability of the physician to determine whether a patient is indigent and Dr. Tudor revised his report as follows:

The Reference Committee reviewed and approved the report of the Committee on Crippled Children, with the reservation that it does not feel that it is practical or advisable for the family physician to screen patients to determine whether or not they are indigent. It feels that the Public Welfare Board should screen these patients instead of the physician. This portion of the report was adopted.

8. *Report of the Committee on Diabetes.* This report was reviewed and approved. This portion of the report was adopted.

9. *Report of the Committee on Foreign Trained Physicians.* This report was reviewed and approved. The Reference Committee suggested that the North Dakota State Medical Association educate the people of North Dakota in regard to the necessity for the Educational Council for Foreign Medical Graduates examinations. This portion of the report was adopted.

10. *The Committee on Liability Insurance.* This committee had no meeting during the year.

11. *Report of the Committee on Maternal and Child Welfare.* This report was reviewed and approved. This portion of the report was adopted.

12. *Report of the Committee on Mental Health.* The report was reviewed and approved. This portion of the report was adopted.

13. *The Committee on Nursing Education.* This committee did not meet during the year.

14. *The Committee on School Health.* This committee did not meet during the year.

15. *Report of the Liaison Committee to the North Dakota State Bar Association.* This report was reviewed and approved. This portion of the report was adopted.

16. *Report of the Liaison Committee to the North Dakota State Dental Association.* This report was reviewed and approved. This portion of the report was adopted.

17. *Report of the Liaison Committee to the State Board of Administration.* This report was reviewed and approved. This portion of the report was adopted.

18. *Report of the Liaison Committee to Blue Cross—Blue Shield.* The Reference Committee suggests that a hospital survey as recommended by this committee be approved. The committee also recommended that this committee be retained for 1960. This portion of the report was adopted.

Dr. R. B. Tudor moved that the report be adopted as a whole. Motion was seconded by Dr. McLean and carried.

R. B. TUDOR, M.D., Chairman

D. W. PALMER, M.D.

WELDE W. FREY, M.D.

R. W. MCLEAN, M.D.

R. E. HANKINS, M.D.

Reference Committee to Consider the Reports of the
Delegate to the AMA,
Committee on Medical Education,
the Representative to the Medical Center
Advisory Council,
and the report of the Representative to the
State Health Planning Committee

Dr. L. G. Pray, chairman of this committee, presented the following reports and their discussions, which were adopted section by section and as a whole.

1. *Report of the Delegate to the AMA.* The Reference Committee read with interest the report of Dr. Willard A. Wright, the delegate to the AMA. Dr. Wright attended all sessions of the House of Delegates during 1959. He serves on 6 committees and is chairman on one of them. There has been considerable concern and activity on the national level with regard to socialized medicine, medical economics, and medical education.

On behalf of the North Dakota State Medical Association, the Reference Committee wished to thank Dr. Wright for his excellent work in the House of Delegates of the American Medical Association. This portion of the report was adopted.

2. *Report of the Committee on Medical Education.* This report was reviewed, and the Reference Committee noted that this report expressed concern over the decline in applicants for admission to the Medical School in 1960. This report recommended that all physicians try to stimulate interest in their own communities in medicine as a career.

The report also mentioned the need for a toxicologist at the Medical School and the efforts being made to obtain one.

Dean Harwood also had reported on the preceptor program and recommended its continuance and improvement. The status of the loan fund was also outlined. The Reference Committee approved of these recommendations. This portion of the report was adopted.

3. *Report of the Representative to the Medical Center Advisory Council.* Dr. Woutat's report described the present status of the Medical School with regard to admissions and enrollment, with special mention of the drop in the number of applicants for admission. The utilization of the Student Loan Fund amounted to \$64,200 in 1959 out of a total of \$75,000 available each year.

Other matters considered in the report were the biochemistry service, the rehabilitation unit, the medical center budget, and the psychiatric training program. This portion of the report was adopted.

4. *Report of the Representative to the State Health Planning Committee.* The Reference Committee reviewed this report and noted that this committee's chief functions are to consider hospital needs in the state and to advise allocations of Hill-Burton federal funds to assist in needed new hospital construction. The present need is mainly for expansion and renovation of present hospitals and for more nursing home facilities. This committee held 2 meetings during the year. This portion of the report was adopted.

Dr. Pray moved that the report be adopted as a whole. Motion was seconded by Dr. Palmer and carried.

L. G. PRAY, M.D., Chairman
J. M. VAN DER LINDE, M.D.
WALTER HANEWALD, M.D.
B. HORDINSKY, M.D.

Reference Committee to Consider the Reports
of the Standing Committees

Dr. Baumgartner, chairman, presented the following reports and their discussions, which were adopted section by section and as a whole.

1. *Report of the Committee on Necrology and Medical History.* The Reference Committee reviewed this report, realizing the great loss the association suffered in the deaths of 9 of its most prominent physicians in the past year: namely, John S. McArdle, Minot; Philip C. Arzt, Jamestown; Frank E. Wheldon, Minot; Donald L. Olson, Fargo; D. J. Halliday, Kenmare; Archie D. McCannel, Minot; Andrew A. Kjelland, Hatton; Oscar T. Benson, Glen Ullin; and J. L. Devine, Minot.

The genius, self-dedication, incredible energy, and unswerving devotion to their ideals has made the life stories of these physicians most inspiring in the annals of modern medicine in North Dakota. Their departure from our midst is most regrettable.

Dr. Baumgartner requested the delegates to show their respect to our departed members by standing a moment in silence. Moment of silence adopted this portion of the report.

2. *Report of the Committee on Public Health.* The committee reviewed and approved the report, and Dr. Baumgartner moved the adoption of this portion of the report.

Dr. Nugent made the following comments at this time: "I note that the Committee on Public Health did not have a formal meeting this past year. Last year the House of Delegates directed the Committee on Public Health to secure the name of a physician to serve as state health officer and to present this name to the governor of North Dakota. As this committee did not meet, evidently it did not follow through this directive and did not submit this name to the governor. In view of this, I feel that this report is not acceptable."

Speaker Dodds next spoke as follows: "You are quite right. That resolution was passed a year ago. The solution to this immediate problem would be to have Dr. Lund appoint a committee that will function and carry out these instructions, as this still stands from last year. All those in favor of accepting the motion? All nays—opposed. The motion failed and the report is not accepted."

3. *Report concerning the proposed Rheumatic Fever Prophylaxis Program.* It was the committee's unanimous opinion that the proposed program should be referred to the Committee on Public Health for additional study and recommendation. This portion of the report was adopted.

4. *Report of the Committee on Legislation.* The Reference Committee reviewed the report and felt that the Committee on Legislation should be commended for its work. It was noted that the next legislative session will again be faced with an increased number of medical bills. It behooves every member of the society to follow such legislation closely. This portion of the report was adopted.

5. *Report of the Committee on Public Relations.* The report was reviewed and approved by the Reference Committee, and Dr. Baumgartner, the chairman, made the following comment: "This Committee on Public Relations did not meet, and I feel very much the same toward it as Dr. Nugent felt regarding the Committee on Public Health. This is one of the committees that should be more active in the future." This portion of the report was adopted.

6. *Report of the Committee on Official Publication.* After reviewing this report, the Reference Committee noted that the committee recommends that THE JOURNAL-LANCET be designated as the official publication for the North Dakota State Medical Association for another three years.

Chairman Baumgartner moved that the association continue THE JOURNAL-LANCET as the official publication for the next three years and also the adoption of this portion of the report. This portion of the report was adopted.

Dr. Baumgartner, chairman of the Reference Committee, moved that the report as a whole be adopted. Motion was seconded by Dr. Palmer and carried.

CARL BAUMGARTNER, M.D., Chairman

C. J. KLEIN, M.D.

W. L. MACAULAY, M.D.

FRANK W. HILL, M.D.

A. F. HAMMARGREN, M.D.

Dr. Boerth, chairman of the Committee on Official Publication, announced that he wished the House to consider a recommendation from his committee.

Dr. Baumgartner stated that the committee recommended that the association be given better recognition on the face of THE JOURNAL-LANCET and that it be stated that it is the official journal of the North Dakota State Medical Association. Perhaps this could be done on the front page or on the contents page.

Dr. Boerth advised the House that this magazine is the official journal for North Dakota only; other states are designated, but they do not feel that this journal is their official publication.

Dr. Hill made the motion that the Committee on Official Publication work out this problem with THE JOURNAL-LANCET concerning the desired changes. Motion was seconded by Dr. Pray and carried.

Reference Committee to Consider the Reports of the Committee on Medical Economics,

Advisory Committee to Public Assistance Division of State Welfare Board, Committee on Rural Health, and Veteran's Medical Service

Dr. James Mahoney, chairman, presented the following reports and their discussions, which were adopted section by section and as a whole.

1. *Report of the Committee on Medical Economics.* The Reference Committee reviewed the report of Dr. Peters, chairman, and highly commended him and his committee for their untiring efforts to produce stability in this difficult field. It recommended that they be allowed to continue their efforts in negotiating with various agencies discussed in this report.

On the advice of the Medical Economics Committee, the Medical Advisory Committee to the Public Welfare

Board has been formed. This committee can be extremely useful to the association. The Reference Committee sincerely desired that the delegates support and cooperate with the committee. It recommended that the report of the Medical Advisory Committee to the Public Welfare Board be sent to each active physician in the state. This portion of the report was adopted.

2. *Report of the Committee on Rural Health.* The Reference Committee commended Dr. Jacobson and his committee. The public demands rural medical care, and the committee is cognizant of the problems involved. As to the suggestion of creating open-staff hospitals, the Reference Committee feels that this proposal poses problems which are impractical at this time and need much further study. It recommended that the Committee on Medical Education and representatives of medical education study the suggestion of rural preceptorship. This portion of the report was adopted.

A discussion followed among several of the delegates concerning open-staff privileges in all hospitals in the state. It was also felt that the smaller communities need specific help along the lines of assistance in obtaining a physician and in determining whether a town has the requirements to attract such a physician. It was the consensus that a committee composed of a few volunteers from the state association could make such a survey to decide either that the town could not support a doctor or that it should have a physician. It was believed that, if this procedure could be developed, the public in rural communities could be educated in regard to their responsibility to the doctors, and public relations in the state could be improved.

Dr. Hankins made the motion that the House of Delegates recommend that all hospitals throughout the state declare open-staff privileges to qualified physicians, subject to local restrictions and limitations. Limitations referred to licensed physicians, subject to hospital staff limitations and restrictions. Motion was seconded by Dr. Mahowald and passed.

Dr. Hankins also moved that the Committee on Rural Health make a survey of the smaller towns seeking a physician. Motion was seconded by Dr. Pray and passed.

3. *Report of the Committee on Veterans Medical Service.* The Reference Committee reviewed this report and commended this committee on its report. This portion of the report was adopted.

Dr. Mahoney, chairman of the Reference Committee, moved that the report as a whole be adopted. Motion was seconded by Dr. Vinje and carried.

J. H. MAHONEY, M.D., Chairman

W. C. DAILEY, M.D.

D. R. STRINDEN, M.D.

EDMUND VINJE, M.D.

(The Transactions will be concluded in the December Issue)

Book Reviews . . .

Measurement of Subjective Responses: Quantitative Effects of Drugs

HENRY K. BEECHER, M.D., 1959. *New York: Oxford University Press.* 494 pages. \$12.75.

The basic purpose of the text is to show that a quantitative approach to sensation, mood, and other subjective responses is possible. The first part of the book provides a comprehensive treatment of the measurement of pain. The second part of the text deals with a quantitative approach to the effects of drugs on a variety of subjective states. Among the subjects discussed are the measurement of mental clouding, sedation and hypnotic states, sleep, the effects of anesthetic agents on subjective states, euphoria and dysphoria, hunger, nausea, itching, and quantifiable expressions of anxiety. Also discussed is a group of drugs now attracting great interest, the psychotomimetic agents.

The book is indexed and will be interesting to those who have the same curiosity as the author.

JOHN S. LUNDY, M.D.
Chicago

Behavioral Change in the Clinic: A Systematic Approach

GERALD R. PASCAL, Ph.D., 1959. *New York: Grune & Stratton.* 124 pages. \$4.75.

This stimulating, slim volume boldly attempts to state systematically some fundamentals about changing behavior—particularly, deviant behavior.

Professor Pascal speaks of over 200 experiments carried out by himself and his students; in 8 of 10 cases, he could boast of altered behavior in the "right" direction.

The logic and arguments reflect frank dependence on a type of behaviorism associated in the 1920's with Watson and are applied to behaviors deviating from some idea of standard behavior. The author postulates that people operate on needs or drives. When stirred by frustrated expectancies, they develop psychologic deficits, shown by deviation from the norm. When needs are met with deprivation, stresses of varied potency occur, which are linked with the business of survival.

These and similar formulations are acknowledged crystallizations from the writings of Freud, Jung, Horney, Murray, Maslow, Lewin, Tolman, Hull, and Hebb.

Factors of habit, environmental situations, and organismic stability are basic in the formulations from which Dr. Pascal derives some wise counsel for his students and others who would like to appraise and manipulate behavior.

The basic variables are given symbolic reference—for example, PD (psychologic deficit), and an effort is made to state functional relationships after the fashion of Kurt Lewin. Whether or not this works out to the reader's advantage depends on such factors as M (memory), Abs. (abstracting ability), and T/A (tolerance/annoyance quotient), to coin a few symbols for the purpose of this review.

My impression is that busy physicians would find this trying, but Professor Pascal's graduate students should not, or they aren't worthy of their bachelor degrees.

However, there are some things stated clearly about psychologic treatment which would make the effort worthwhile even for the busy physician, some formulations that should prod experienced practitioners in this field, and some suggestive results from small experiments to prompt the experimentally minded. There is also a good bibliography at the end of each chapter.

JOHN ARSENIAN, Ph.D.
Boston

Diagnostic Radioisotopes

CHARLES A. OWEN, JR., M.D., 1959. *Springfield, Ill.: Charles C Thomas.* 381 pages. Illustrated. \$15.75.

This book separates diagnostic from therapeutic radioisotope procedures and places them in the realm of the clinical laboratory. A disagreement about such separation from the radiologic departments will be voiced by many radiologists interested in diagnostic isotope procedures. However, as the author says, a clinical laboratory already has a good basic background of analysis and technique which can be readily adapted to diagnostic isotope methods. The presentation is mainly that of the clinical pathologist and is directed to the laboratory study of diseases that reveal themselves readily to isotopic study and detection. Very little clinical material is presented in this book.

The author adheres to his intent to make this book a laboratory guide and to present a review of the physiologic principles and techniques of diagnostic radioisotope procedures. The sections on metabolic studies using isotopes, especially radioiodine, are quite complete and clearly presented. Similarly, the section on hematology is particularly clear and well outlined. The less well-established and newer diagnostic procedures are briefly outlined and evaluated.

ROY R. GREENING, M.D.
Philadelphia

Diagnosis and Treatment of Diseases of the Trachea and Bronchi

HERMAN J. MOERSCH, M.D., and HOWARD A. ANDERSEN, M.D., 1960. *Springfield, Ill.: Charles C Thomas.* 108 pages. Illustrated. \$4.25.

It has been said that the physician who sees patients does not have time to write books. This criticism cannot be leveled at the authors of this little monograph, however, which contains within its small bulk the observations and mature advice of two men whose experience in the joint fields of diseases of the chest and endoscopy is at the same time practical and extensive. Although some of the diseases mentioned border on the exotic, a proper balance is struck between the smaller space devoted to them and the larger sections on the more commonplace problems of daily practice. It is an entirely practical manual, and theoretic discussion is limited to a refreshing minimum. Where necessary, conflicting opinions on management are paid due attention without leaving the reader in doubt as to the opinions of the authors.

A few minor publishing errors unfortunately have been allowed to creep into the text. The table on tracheal tumors, for example, is found in the section on tracheal trauma. No doubt the authors will wish to correct the statement that "dilatation of the left ventricle as in mitral stenosis may give rise to bronchial obstruction which always involves the left main stem bronchus." These small discrepancies can be taken care of in subsequent editions. The practicing thoracic physician and the endoscopist will find this a worthwhile addition to their medical bookshelves.

MATTHEW B. DIVERTIE, M.D.,
Rochester, Minnesota

Instrumentation in Anesthesiology

WILLIAM H. L. DORNETTE, M.D., 1959. Philadelphia: Lea & Febiger. 242 pages. Illustrated. \$8.00.

In an era when poor-risk patients are being subjected to more complex surgical procedures, electronic monitoring of physiologic phenomena during surgery is becoming commonplace. The purpose of this book is to explain the principles of operation as well as the application of monitoring devices. In addition, techniques commonly employed in anesthesiology research laboratories are considered in some detail. The information contained herein is also intended to serve as a guide for the prospective purchaser of apparatus for the operating room or for the laboratory. There is a great need for a text to aid the investigator and the clinician in the understanding, critical evaluation, and use of instruments. Unfortunately, experience and knowledge among anesthesiologists may vary from a vague recollection of high school physics to the ability to design or apply new instruments. To bridge the gap with a single text is impossible.

The major weakness of this work is its failure to describe some very popular and very excellent equipment available to the anesthesiologist. In most cases, the equipment described is representative; however, this is not universally true. The space devoted to electrocardiographic interpretation would have been better spent in an evaluation of commonly employed operating room cardioscopes and cardiac monitors. The oxymograph described is by no means the most popular on the market, nor is it representative of the available instruments. The description of the infrared carbon dioxide analyzer is excellent, but no consideration is given to any of the new, improved analyzers. The microgasometer is not mentioned, even though it is replacing the Van Slyke apparatus in cardiorespiratory laboratories. Gas chromatography is treated superficially; however, as the authors point out, this technique will soon replace all manometric and volumetric methods for the analysis of blood, respired gases, and anesthetic agents.

In spite of these shortcomings, the book serves a most useful purpose. The initial chapter provides an excellent introduction for the neophyte into the world of basic physics as applied to electronics. The chapters on amplifiers and recorders are equally well done. The discussion of principles and techniques of operation of the apparatus presented is excellent. The bibliography at the end of each of the 21 chapters provides a good starting point for those with specific problems or interests. The paper and printing are excellent, the index adequate, and the illustrations abundant.

This work should be of real value to anesthesiologists not conversant with the principles of electronic monitoring devices. It provides a basic fund of information for

those interested in analysis of blood and respired gases. It should be required reading for all physicians training in anesthesiology. The experienced research worker, however, will find little of value. *Instrumentation in Anesthesiology* should be in the library of every department of anesthesiology.

HOWARD L. ZAUDER, M.D.,
New York City

Haemorrhagic Nephroso-Nephritis

A. A. SMORODINTSEV, V. G. CHUDAKOV, and A. V. CHURILOV; translated from the original Russian by CATHERINE MATTHEWS, 1959. New York: Pergamon Press. 124 pages. Illustrated. \$9.00.

This monograph, translated from the Russian language, is concerned with an unusual and fascinating disease very similar to acute hemorrhagic fever. The authors, who are highly regarded by virologists, present considerable experimental data to support their beliefs but do not prove that this is a viral infection. Considerable detail is given to the territorial distribution, seasonal incidence, and proposed method of transmission of the disease. The prophylactic measures are well described.

The clinical course—its general characteristics and stages of development—is succinctly pictured and correlated with the pathologic anatomy. Nine pages of charts are of considerable aid in the differential diagnosis. The treatment is primarily supportive in nature. However, conspicuous by its absence is the failure to describe the conservative treatment of acute renal failure and dialysis with the artificial kidney.

The text is well written—or at least well translated from the Russian language. The illustrations are few and are of fair quality. The bibliography is good. This is primarily a disease of Russia and the Far East, and, for this reason, the text is of value only to the epidemiologist, virologist, and physician who practices in this limited geographic area.

MILTON P. REISER, M.D.,
Minneapolis

The Truth About Your Eyes

DERRICK VAIL, M.D., 1959. Second edition. New York: Farrar, Straus, and Cudahy, Inc. 180 pages. Illustrated. \$3.50.

This little book contains a world of information about the eye. It was primarily written for laymen who are interested in teaching sight-saving classes and hygiene and for those who teach the blind. It is recommended reading for the personnel of our State Aid to the Blind program.

This book discusses with scientific authority the eye and vision and tells what we should know about the care and safeguarding of our eyesight. It debunks many misconceptions held by the public on this vital subject.

Dr. Vail starts his book in the first 4 chapters with a brief but accurate description of the development, anatomy, and function of the eyes and their adnexa. This is written in such a way as to be grasped and appreciated by the average person.

In Chapter 5, refraction and the different types of refractive errors are discussed, as is the need for their correction. Several erroneous ideas concerning glasses are brought out. Also in this chapter we find the value

(Continued on page 30A)

Blood pressure that goes up with stress often comes down with SERPASIL®

(reserpine CIBA)

One reason that many cases of hypertension respond to Serpasil is that many cases are associated with stress. Stress situations produce stimuli which pass through the sympathetic nerves, constricting blood vessels, and increasing heart rate. Hyperactivity of the sympathetic nervous system may elevate blood pressure; if prolonged, this may produce frank hypertension. By blocking the flow of excessive stimuli to the sympathetic nervous system, Serpasil guards against stress-induced vasoconstriction, brings blood pressure down slowly and gently.

*Coan, J. P., McAlpine, J. C., and Boone, J. A.: J. South Carolina M. A. 51:417 (Dec.) 1955.

72930MB

Complete information available on request.

In mild to moderate hypertension, Serpasil is basic therapy, effective alone "...in about 70 per cent of cases..."*

In severe hypertension, Serpasil is valuable as a primer. By adjusting the patient to the physiologic setting of lower pressure, it smooths the way for more potent antihypertensives.

In all grades of hypertension, Serpasil may be used as a background agent. By permitting lower dosage of more potent antihypertensives, Serpasil minimizes the incidence and severity of their side effects.

C I B A
SUMMIT, N. J.



BOOK REVIEWS

(Continued from page 572)

of eye exercises, visual training, and optical aid for those of low visual acuity which are subjects of interest to those teaching handicapped children. I heartily agree with statements about tinted and colored glasses, which are nowadays worn by many and needed by a very few.

A short chapter on muscular disturbances and squint should be read by every mother who has a child with an eye deviation and would give her a better knowledge of this distressing abnormality and the means taken for its correction.

There are short, interesting chapters on vitamins and the eyes, corneal transplantation, cataracts, and diseases affecting vision in infancy. A very important and well-written chapter is that on glaucoma, the disease that causes more blindness than any other. The knowledge of the prodromal signs and symptoms of this dreaded disease should be known by everyone.

Dr. Vail discusses other common diseases of the external eyes as well as internal diseases of the eye and what to do for first aid in injuries to the eyes or eyelids.

The last chapter, "The Hygiene of Vision," is very interesting. Dr. Vail talks about good reading habits, television and eye strain, and eye strain and glasses. He gives 5 recommendations for television viewing. He closes this chapter and the book with 10 rules for good eye health that everyone should know and follow.

I recommend that nurses, medical students, and general practitioners with little knowledge of the eye sit down for an hour or so and read this little book. I am sure they will find useful knowledge about the eye, and their time will be well spent.

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Bismarck, North Dakota

Acute Cardiac Pulmonary Edema

SIGMUND WASSERMAN, M.D., 1959. *Springfield, Ill.: Charles C Thomas.* 101 pages. \$4.25.

The title of this monograph is somewhat misleading. Its text is a highly personalized presentation of the author's ideas on the neurogenic induction of acute cardiac pulmonary edema rather than a comprehensive discussion of the subject.

The author presents solid evidence for the widely accepted reflex precipitation—via "hyperexcitable" CNS centers—of acute pulmonary edema (sudden onset, rapid relief by morphine, psychogenic precipitation, and so on). However, he uses his own observation of the very occasional improvement following carotid sinus stimulation as the focal point of the entire book. He builds an inverted pyramid with this finding at its apex to support the existence of aortic, carotid, and coronary receptors which are said to initiate the pulmonary edema reflex arc. The existence of low-grade pulmonary congestion between attacks and consequent possibility of pulmonary receptors are not considered. No statistical studies are presented, and the admission that "the percentage of pulmonary edema cases which respond to carotid sinus pressure is very low for any observer" is dismissed since the process is so "complex" that "the negative responses are paled into insignificance." While this may hold, it is not adequately supported in the ensuing discussion.

The example of pulmonary edema following cerebral lesions is used without mention that, in well-studied series, virtually every such patient has had significant heart damage. The discussion of treatment is sketchy,

and its summary emphasizes the use of carotid sinus pressure and goes on to list morphine but places "tepid hand and foot baths" and "hot mustard over the precordium" ahead of oxygen and digitalis. This is in line with the author's concepts but will not find wide acceptance as a practical guide to action.

The major fault of this book lies in its one-sided presentation of a very complex subject. The accuracy of the clinical observations and faithfulness of the case reports cannot be questioned. Yet the conclusions drawn (many quite reasonable) are not supported in a convincing manner. An excess of *ex cathedra* quotation ("Wenckebach opined") is employed with no orderly marshalling of data. It is entirely possible that the reader could reach the same conclusion upon studying such references, but efficient quotations would obviate the need to do so before passing to the next point. Hand in hand with this, but somewhat less detrimental, is the author's literary style, which, although pleasant and readily readable, mars the presentation by repetitiousness and the absence of clear definition of terms.

Although the book was published in 1959, relatively few of the 166 references and numerous footnotes quote work published after 1930. A "new clinical concept" dates from 1930 (page 10). "Syphilitic aortitis" is repeatedly used as an example. It is cited as one of the 3 principal causes of acute pulmonary edema with no indication of the era during which this may have been true.

Among the pages of this monograph one can yet find much evidence of an inquiring mind and of much fruitful investigation. The poor impression given by the text does not eclipse the author's past contributions to medical theory and practice. Yet the information of value could have been presented in one-half the number of pages, even with a balanced discussion and a few diagrams. A less general title would have been more descriptive.

The book is well bound and clearly printed on paper adequate for a volume devoid of illustrations. The table of contents is fairly comprehensive, but there is no index. A list of Dr. Wasserman's many publications is appended. The unsigned preface suggests that this publication was instigated as a personal tribute by his many admirers. In this sense it does not lack charm and as a "period piece" testifies to the author's undoubted greatness. As such, however, it must be purchased as a luxury item. It will irritate cardiac physiologists, provide unlimited targets for sniping statisticians, confuse indiscriminating students, and provide little of usefulness for general or specialized practitioners.

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EDITOR'S NOTE:

In the article "Intravenous Angiography" by Samuel F. Feinberg, M.D., and Marvin E. Goldberg, M.D., which appeared in the September 1960 issue of *THE JOURNAL-LANCET*, the sentence on page 417, paragraph 1, reading, "The volume used ranges from approximately 1 to 100 cc. per kilogram," is incorrect. It should read: "The dosage used is approximately 1 cc. per kilogram. The range of dosage is from 70 to 100 cc."

On page 334 of the article "Neonatal Intestinal Obstruction" by Robert H. Whittlesey, M.D., which appeared in the July 1960 issue, the outlines for tables 2 and 3 should be transposed.

The Journal Lancer

SERVING THE MEDICAL PROFESSION OF MINNESOTA,
NORTH DAKOTA, SOUTH DAKOTA, AND MONTANA

Cesarean Section A Ten-Year Analysis of its Use in a Rural Community

ROBERT E. LUCY, M.D.

Jamestown, North Dakota

FOR THE PAST several years, one of the major activities of the North Dakota Society of Obstetrics and Gynecology has been an intensive maternal mortality study, undertaken in a sincere effort to prevent or reduce the incidence of maternal mortality within our state, even though the present incidence is low in comparison with studies undertaken elsewhere. As a corollary to this activity and since no such study has been made in my community, I analyzed the indications for cesarean section and the results of its use in my community. I found this study to be both stimulating and revealing.

The material for this paper has been taken from 2 hospitals with a combined approximate capacity of 200 beds located in a community of 15,000 and embracing a practice area of approximately 50,000. The majority of the work in obstetrics is done by 3 physicians, with occasional deliveries being done by 3 others. Of these, 1 man limits his practice to obstetrics and the others are general practitioners. It was with the cooperation of record room personnel that I

was able to review the charts of all patients undergoing cesarean sections during the years 1949 through 1958.

INCIDENCE

In the ten-year period, there were 7,552 deliveries. One hospital had 3,789 with 80 sections, or an average of 2.1 per cent, and the other hospital had 3,763 with 116 sections, or 3.1 per cent. A number of these patients were not originally seen by the attending men in town but were brought in from the surrounding areas in difficulty. A total of 196 cesarean sections were performed during this period, or a percentage of 2.6. Of this total, 115 were performed by 1 obstetrician. The others were performed by 7 general surgeons.

PREOPERATIVE DIAGNOSIS AND INDICATIONS FOR CESAREAN SECTION

The innumerable indications for these cesarean sections were divided into the following 9 major conditions, as classified by Bryant¹ in his article in the *American Journal of Obstetrics and Gynecology* in March 1956:

1. Mechanical obstruction to delivery, including all cases of fetal-pelvic disproportion and malpresentation and malposition.

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This paper is the presidential address presented at the meeting of the North Dakota Society of Obstetrics & Gynecology, Grand Forks, September 1959.

TABLE 1
TYPES OF SECTIONS

Year	Primary			Secondary			Total sections	Total deliveries	Per cent
	Total	Low Cervical	Classical	Total	Low Cervical	Classical			
1949	13	10	3	7	3	4	20	818	2.4
1950	7	2	5	7	3	4	14	750	1.9
1951	12	7	5	13	6	7	25	810	3.1
1952	11	8	3	7	5	2	18	829	2.2
1953	10	6	4	4	3	1	11	721	1.9
1954	14	12	2	7	6	1	21	713	2.9
1955	15	13	2	10	9	1	25	719	3.5
1956	7	6	1	12	11	1	19	721	2.6
1957	8	6	2	12	11	1	20	710	2.8
1958	7	6	1	13	10	3	20	761	2.6
Total	104	76	28	92	67	25	196	7,552	2.6
	53.1%	73.1%	26.9%	46.9%	72.8%	27.2%			

Per cent of low cervical to total sections 72.9
Per cent of classical to total sections 27.1

2. Bleeding, including placenta previa and premature separation of the placenta.

3. Hypertensive disease, including preeclampsia, essential hypertension, and eclampsia.

4. Fetal indications, such as fetal distress.

5. Functional anomalies, consisting of prolonged labor alone, with inertia, or with fetal distress and uterine inertia alone.

6. Abnormalities of the reproductive organs, including congenital cloaca and malformations acquired by previous perineal or uterine surgery.

7. Miscellaneous, including previous obstetric difficulties, breech presentation in elderly primiparas, and patient's request for cesarean section.

8. Concurrent or intercurrent diseases, such as simple schizophrenia and diabetes alone or with preeclampsia.

9. Repeat cesarean sections.

TYPES OF SECTIONS

Of the total number of sections, 53.1 per cent were primary and 46.9 per cent were repeat. Of the total primary sections, 73.1 per cent were low cervical and 26.9 per cent were classical. Of the repeat sections, 72.8 per cent were low cervical and 27.2 per cent were classical. In the total series, 72.9 per cent were low cervical and 27.1 per cent were classical (table 1). Over the past few years, the incidence of the low cervical section has gradually increased. Of all the sections that were done, 52, or 26.5 per cent, were on primiparas, and 144, or 73.5 per cent, on multiparas. All primiparas sectioned in our area had a repeat section. Some of the multiparas

TABLE 2
INDICATIONS FOR CESAREAN SECTIONS

	Primipara	Multipara
Prolonged labor	15	
Previous cesarean section		92
Placenta previa	1	19
Abortion	5	4
Cephalopelvic disproportion		
Hydrocephalic	1	2
Small pelvis	16	1
Transverse lie	1	6
Eclampsia	3	
Brow presentation		1
Face presentation		1
Previous obstetrical difficulty		6
Previous perineal surgery		3
Uterine inertia	3	2
Fetal distress	1	
Simple schizophrenia		1
Elderly primipara with breech	1	
Cloaca		1
Diabetes preeclampsia	1	
Diabetes		1
Previous uterine surgery	1	
Twin pregnancy, unable to deliver	1	
Severe preeclampsia with prolonged labor	1	
Prolonged labor with fetal distress		1
Prolonged labor with inertia	1	
Hypertensive disease		1
Patient's request		2
Total	52	144
	26.5%	73.5%

were allowed to deliver vaginally, depending on the circumstances of their first section.

The most frequent indications for the primary sections were mechanical obstruction, bleeding, and functional anomalies (table 2). A further analysis of this group reveals that functional anomalies accounted for 11.2 per cent, bleeding for 14.7 per cent, and mechanical obstruction for 13.3 per cent. Repeat sections totaled 92, or 46.9 per cent and accounted for a little over 63 per cent of all the sections that were done on multiparas.

Of all the sections that were done for bleeding, 65.5 per cent were for placenta previa, which occurred in multiparas in all but 1 case. Premature separation of the placenta was seen 9 times—5 in primiparas and 4 in multiparas. No maternal deaths due to bleeding were recorded.

ANESTHESIA

In the past, most of our patients were sectioned under spinal anesthesia. Recently, I have been using cyclopropane with nitrous oxide, oxygen, and succinylcholine. The patient is induced with nitrous oxide and oxygen. Cyclopropane is added after thirty seconds and increased fairly rapidly to 1,000 cc. Succinylcholine, 2 mg. per cubic centimeter, is started slowly at 20 drops per minute. Cyclopropane is reduced to 100 cc. per minute after the patient is lightly anesthetized. This is usually adequate to hold the patient throughout surgery. In over 90 per cent of the cases, the infant responded immediately. The mother usually responded by the time surgery was completed.

STERILIZATION

In one hospital, the section rate was somewhat higher because its regulations permit steriliza-

tion after repeat sections or for other indications. Until four years ago, no consultation was required for sterilization. At that time, a committee of 3 physicians was appointed to review each case and then decide on the merits of each whether sterilization was indicated. During our period of study, 35 patients, or 17.8 per cent, were sterilized. The ages of the patients ranged from 19 to 42 years, with an average age of between 32 and 33. There were 8 sterilizations performed on gravida II, 12 on gravida III, 8 on gravida IV, and the rest were on gravida V to IX patients. Of the type of sections, 25 were classical operations carried out with the sterilization, and 10 were low cervicals. A few incidental procedures were also done at the time of cesareans, such as salpingo-oophorectomy, repair of incisional or umbilical hernias, and vein ligation and stripping. In the majority of patients, 25 of the 35—sterilization was carried out because of previous cesarean sections; in 2, because of placenta previa and the fact that the patients were grand multiparas; and, in the rest, because of other indications, such patient's request, recent perineal repair, previous obstetric difficulties, diabetes, transverse lie, and hypertension (table 3).

TRANSFUSION

Until recently, the only blood that has been available in the hospitals has been from a walking blood bank or a few units that the hospitals had collected. A short time ago, we joined the Southwest Blood Bank System, so that it is no longer a problem to provide adequate blood replacement. Fortunately, we have always been able to secure all the blood that has been needed for surgery, and, during this period of study, 41 patients were transfused either during, before, or after surgery. The amount of blood required varied from 1 to 6 pt.

TABLE 3
STERILIZATION

Age groups	No. patients	Gravida	No. patients	REASONS FOR STERILIZATION	No.
15 to 20	1	II	8	Previous cesarean sections	25
21 to 25	4	III	12	Placenta previa	2
26 to 30	9	IV	8	Mental	1
31 to 35	7	V	2	Transverse lie	1
36 to 40	11	VI	1	Hypertension	1
41 and above	3	VII	1	Recent perineal repair	1
		VIII	2	Patient's request	2
		IX	1	Previous obstetric difficulty	1
				Diabetes	1

Average age: 32.6
17.8% patients sterilized during cesarean section

MORBIDITY

It is difficult to determine the incidence of morbidity because a number of these patients were routinely placed on antibiotics postoperatively. Only 1 patient in this series experienced much difficulty and she was a gravida IV with a small bowel obstruction. She had had 3 previous vaginal deliveries, and, in the fourth pregnancy, she had had a transverse lie presentation that could not be manipulated to a cephalic or breech position and had to be sectioned.

CAUSES OF INFANT DEATHS

During the ten years under study, there were 7,356 vaginal deliveries, with an infant death rate of 229, or 3.1 per cent (table 4). The highest death rate was 40.6 per cent in the premature group, and the next highest was 38.4 per cent in the stillborn group. Congenital malformations accounted for 9.6 per cent of the deaths, erythroblastosis fetalis for 6.6 per cent, and atelectasis for 4.8 per cent.

In the group of patients undergoing cesarean sections during the same period, there were 21 infant deaths in 196 cesarean sections, or 10.7 per cent. Stillbirths constituted the largest percentage of these deaths, of which there were 8, or 38.1 per cent. In this group with stillborn infants, the fetus was in a transverse lie position in 2 and could not be delivered from below; 4 were in the bleeding category; 3 with premature separation of the placenta and 1 with placenta previa; 1 was a diabetic with a superimposed pre-eclampsia which didn't respond to treatment; and 1 had an induction with subsequent fetal distress. Deaths in the premature group were next in frequency, numbering 4, or 19 per cent. Of these, 2 had elective cesarean sections performed by general surgeons, and

the babies weighed between 3 and 3.5 lb., respectively, at the time of delivery and expired. Why these patients were sectioned at that time, I was unable to determine. Whether a "waiting to labor" policy in these cases would have resulted in the survival of the baby or whether it would have increased the risk for the mother, since both of these patients had had previous sections, is a question that cannot be answered. Erythroblastosis caused the death of 2 infants in this series, both delivered by repeat cesarean sections. One infant died of pneumonia on the third postpartum day, 1 of hyaline membrane disease, and 2 of atelectasis. Three babies died of congenital malformations, all of whom were hydrocephalic infants, and the indication for section was cephalopelvic disproportion. It was quite interesting that 2 of these infants showed congenital toxoplasmosis on autopsy. Both of these cases have had slides sent to several different centers to affirm the diagnosis. One patient had a titer of 1 to 1,050. We lost track of the other patient and were unable to obtain a titer.

To compare the fetal mortality according to section indication (table 5), there were 5 deaths in the repeat section group, or 23.8 per cent. Now, of this group, 2 infants were the previously mentioned prematures, 1 died of hyaline membrane disease, and 2 were erythroblastotic. In the groups with cephalopelvic disproportion due to fetal obstruction, all 3 infants were hydrocephalic. The death rate in this group was 14.3 per cent. In the group with small pelvis, 8 patients were sectioned because of the findings on x-ray pelvimetry. In other patients, failure of the head to descend after twenty-four to seventy-two hours was the indication for cesarean section. In the bleeding category, placenta previa was

TABLE 4
MAJOR CAUSES OF INFANT DEATH IN
7,356 VAGINAL DELIVERIES AND 196 CESAREAN SECTIONS

Causes	Vaginal deliveries		Cesarean sections	
	Number of deaths	Per cent	Number of deaths	Per cent
Prematurity	93	40.6	4	19
Stillborn	88	38.4	8	38.1
Congenital malformations	22	9.6	3	14.3
Erythroblastosis	15	6.6	2	9.5
Atelectasis	11	4.8	2	9.5
Pneumonia			1	4.8
Hyaline membrane			1	4.8
Total	229	3.1	21	10.7

TABLE 5
FETAL MORTALITY ACCORDING TO SECTION INDICATION

	Number of deaths	Per cent of fetal deaths	Per cent according to total number of sections
Previous section	5	23.8	2.55
Cephalopelvic disproportion	3	14.3	1.53
Bleeding			
Premature separation	5	23.8	2.55
Placenta previa	3	14.3	1.53
Prolonged labor and preeclampsia	1	4.8	.51
Diabetes and preeclampsia	1	4.8	.51
Transverse lie	2	9.5	1.02
Fetal distress			
Failure of induction	1	4.8	.51

present in 3 patients, or 14.3 per cent, and premature separation of the placenta occurred in 5, or 23.8 per cent. One death was due to prolonged labor and preeclampsia, 1 to diabetes and preeclampsia, and 1 to fetal distress following induced labor. Of the fetal deaths, 2, or 9.5 per cent, were in patients who had had cesarean sections because of the transverse lie of the fetus.

MATERNAL MORTALITY

Hess² reviewed the literature and found a maternal mortality rate of 0.05 to 0.3 or 0.4 per cent and also found several authors with a series of more than 1,000 sections without a maternal death. The nationwide mortality rate is about .52 per cent which is probably 2 to 4 times that associated with vaginal deliveries. There were no maternal deaths in our series.

COMMENTS

During Powell and associates³ eleven-year study, their cesarean section rate was 2.4 per cent and they noticed a slight increase in the last few years. They found that the average rate over the entire country was about 4.2 per cent. They felt that this slight increase in cesarean sections through the years was justified but that, if the incidence exceeded 6 to 7 per cent it was being overdone. We had a repeat cesarean section rate of 46.9 per cent, which compares quite favorably with the rates of other reported series varying between 12.1 and 43.6 per cent. Powell and associates³ reported an incidence of 43.6 per cent.

Our series contained 20 cases of cephalopelvic disproportion, or 10.2 per cent. The series collected by Powell and co-workers³ varied between 13.8 to 53.9 per cent. We had 3 deaths in this series of 20 cases, all occurring in hydrocephalic

infants, 2 of whom had toxoplasmosis. We had 21 fetal deaths in our entire series of 196 cesarean sections, a gross incidence of 10.7 per cent. According to Powell and associates,³ various authors have reported an incidence from their collected series of 4.5 to 8.8 per cent. McNeal's⁴ series varied from 3.7 to 13.5 per cent. Bryant's¹ fetal loss was 7.6 per cent. In our total fetal loss, we had 2 deaths in premature infants that could have been prevented had more care been taken in checking these patients preoperatively. Hess² found an infant mortality of 0 in a series of 422 uncomplicated cesarean sections. He concluded that repeat or elective sections per se offer little risk to the infant. In one of our cases of placenta previa, death of the infant might have been prevented had the attending physician brought the patient into the hospital instead of trying to treat her in a small maternity home with different types of so-called coagulant drugs.

Of the stillborn group, possibly 1 death would have been prevented had there been less delay between the time a transverse lie presentation was diagnosed and the time required to call a surgeon to perform the cesarean section. Attempted version also failed in the other patient with a transverse lie presentation and, by the time the cesarean section was done, it was too late. The 1 mother with placenta previa whose infant was stillborn was not operated upon for the sake of the baby but in order to save her. One diabetic patient with severe preeclampsia should have undergone a cesarean section earlier, but, due to a disagreement about the evaluation of fetal viability on the roentgenogram, operation was delayed. The baby weighing well over 5 lb. when the procedure was performed, expired. She was sectioned not to empty the uterus per se but because she failed to respond to treat-

ment for this superimposed preeclampsia and her general condition was becoming worse.

Of the patients with premature separation of the placenta who delivered stillborn infants, 2 were sectioned because of the mother's condition, and the other entered the hospital with a diagnosis of premature separation after a careful vaginal examination. Since the latter patient was beginning to dilate, an attempt was made to induce labor by administering intravenous Pitocin, and, during this time, the baby's condition became suddenly worse. In an effort to save the baby, the mother was sectioned but delivered a stillborn.

Cesarean sections were performed in 2 patients for abruptio placenta, 1 at twenty-six weeks because of the mother's condition, the baby living 24 hours and the other at eight months, the baby living less than 24 hours. I feel that the latter death could have been prevented, since I am sure that the care in the nursery was not as it should have been. One patient with placenta previa had a cesarean section and delivered twins, but the babies lived only 24 hours.

SUMMARY

1. A review has been made of 196 cesarean sections which were performed in 7,552 deliveries in a rural community.
2. No maternal deaths were recorded in this series.
3. Comparing our statistics with larger institutions shows that good obstetrics can be practiced in small communities.
4. An improvement in obstetric care throughout the state might result if similar studies were undertaken within the several other major communities of our state, since any analysis of obstetric practice tends to reveal areas in which improvement can be achieved.

The author wishes to thank John A. Swenson, M.D., of the DePuy-Sorkness Clinic for his assistance in helping to prepare this manuscript.

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EXPECTORATION OF BLOOD or blood-stained sputum is not always due to pulmonary tuberculosis or bronchial carcinoma. Often, no cause is detected. The incidence of hemoptysis as an initial symptom of active pulmonary tuberculosis has declined, but such bleeding is still a frequent and important symptom of bronchial carcinoma.

In patients with carcinoma, hemoptysis tends to be slight but usually lasts two weeks or more. With active tuberculosis and bronchiectasis, bleeding is brisk and of short duration. A single expectoration of blood-streaked sputum is common for patients with chronic bronchitis or quiescent pulmonary tuberculosis.

Bronchoscopic examination is recommended in addition to roentgenographic and bacteriologic studies only when hemoptysis is certain and repeated or for men past 40 years of age, because of the possibility of cancer.

No abnormality or only upper respiratory infection was found in 44 per cent of 324 patients with hemoptysis. Acute inflammation of bronchi, chronic bronchitis, and bronchiectasis accounted for 30 per cent of cases, but bronchial carcinoma and active pulmonary tuberculosis each accounted for only 4 per cent. After two years' study of 273 patients, the original diagnosis was changed in only 3 instances. Each of the 3 patients, among 194 with normal chest films, was found to have carcinoma.

R. N. JOHNSTON, W. LOCKHART, R. T. RITCHIE, and D. H. SMITH: Hemoptysis. *Brit. M. J.* 5173: 592-595, 1960.

Current Status of Tranquilizing and Antidepressive Drugs

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BURTRUM C. SCHIELE, M.D.

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THE RAPID INTRODUCTION of psychotherapeutic drugs has presented great problems for the general practitioner. Never before has the physician been faced with such a voluminous literature of recently acquired information. Never before have so many new drugs been introduced in such a short time. The understanding of concepts alone has been difficult to achieve. Added to this are the vagaries created by relatively poor understanding of the disorders for which the drugs are intended. The deceptive action of the placebo and the play of the mind also have contributed to present concern about the usefulness of drugs intended to treat disturbances of mood and behavior.

Although questions may be raised about the efficacy of many of the introductions, the general utility of the drugs has now been well-documented. The correlated experiences between drug utilization and the decrease in restraints and resident populations of the New York State Hospital, as cited by Brill and Patton,^{1,2} have been confirmed by those in other states and countries.³ In spite of the ever-increasing number of new admissions to psychiatric institutions, the discharge rate of patients (particularly schizophrenics) has exceeded the number of admissions each year since 1955—the year when the drugs came into use.

Because of this background, great interest exists in these drugs—in current theories of their nature, in their mechanisms of action, and in their similarities and differences. An organized presentation of the available information, therefore, should aid the physician in his orientation to therapy.

In general, psychopharmacologic agents include drugs that act on the nervous system di-

rectly or indirectly to affect mental and emotional processes. Three main classes of agents are of interest: the tranquilizers, the antidepressants, and the psychotomimetics. The latter class refers to agents now undergoing research investigation; they will not be reviewed in this paper. Only agents of current psychotherapeutic interest which fall into the tranquilizer and antidepressant classes will be discussed.

THE TRANQUILIZERS

Tranquilizers are drugs which are used to control anxiety, psychomotor agitation, and related symptoms. As suggested by Schiele and Benson,⁴ they consist essentially of 2 clinical groups: (1) the major tranquilizers and (2) the minor tranquilizers. Major tranquilizers may be divided into the phenothiazines and rauwolfia derivatives, and minor tranquilizers into the substituted diols, the diphenylmethanes, and a miscellaneous group. Additional categories are becoming necessary as new compounds appear. Although this classification is incomplete and tentative, it currently offers the practicing physician a useful guide. The distinguishing clinical features of the major and minor groups presented by the authors⁴ several years ago remain essentially valid.

The major tranquilizers are characterized by the following points:

1. These drugs produce a type of emotional calmness with relatively little sedation; they have proved useful in controlling the symptoms of acutely and chronically disturbed psychotic patients.

2. They are capable of producing the reversible extrapyramidal syndrome characterized by rigidity, tremors, and drooling.

3. The incidence of annoying side reactions is relatively high with the use of these drugs, and serious dangers do exist to some extent.

4. They produce little, if any, dependency or habituation.

The minor tranquilizing drugs are characterized by other points:

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TABLE 1
PHARMACOLOGIC EFFECTS OF TRANQUILIZERS

Type of action	Majors		Minors	
	Phenothiazines (chlorpromazine)	Rauwolfias (reserpine)	Diphenylmethanes (benactyzine)	Substituted diols (meproamate)
Response to afferent stimulation	—	—	—	—
Conditioned reflexes	—	—	—	0
Effect on posterior hypothalamus	—	—	—	—
Blood pressure	—	—	0	0
Body temperature	—	—	0	0
Pupillary size	0	—	0	0
Intestinal activity	0	+	—	0
Antihistaminic activity	+	0	+	0
Adrenergic antagonism	+	+	0	0
Effect on muscle tension	0	0	0	—
Occurrence of tremors	+	+	0	0
Tendency for seizures	+	+	+	—
Effect on rhinencephalic system	+	+	U	—

(0) No significant effect
(+) Increased

(U) Unknown
(—) Decreased

1. These drugs produce a type of calmness or relaxation, but not of the same quality as that produced by the major tranquilizers. These drugs may have a mild sedative effect but are not highly effective in treating disturbed psychotic patients. They are useful in the treatment of psychoneurotic problems and common nervous tension.

2. They do not produce the extrapyramidal motor phenomena so characteristic of the major tranquilizers.

3. The incidence of annoying side reactions with the use of these drugs is relatively low. Dangerous reactions are rare.

4. Habituation may occur with the use of some of these agents.

Pharmacologically, there are distinctions of a somewhat different order among the tranquilizers. Table 1 shows that, although many similarities exist in the actions of representative drugs from each group, there are also considerable differences. In general, the major tranquilizers exert greater autonomic effects and, in addition, are prone to induce tremors. Their most significant action is their ability to depress the hypothalamus without depressing the cortex.⁵ This action may be related to their ability to stimulate the rhinencephalic system—an action which is not shared by the barbiturates and meproamate. The minor tranquilizers differ among themselves in that autonomic blocking activity is more characteristic of the diphenylmethanes; muscle relaxant action is a prominent effect of the substituted diol and miscellaneous groups.

MECHANISMS OF ACTION OF THE TRANQUILIZERS

Whereas the mechanisms of action of the tranquilizers remain in doubt, a good deal of information is available about their central effects. This is particularly true in the case of the major or true tranquilizers. Although both the phenothiazine and rauwolfia groups have essentially the same marked effect against psychomotor excitation, their pharmacologic actions in achieving the effect are quite different. Compounds of both groups are capable of reducing sympathetic excitation at the central level, yet each does this in a different manner. To better understand these actions of normal physiologic responses, one may refer to the publications of Magoun and associates.⁶⁻⁸

Chlorpromazine—the most widely studied of the phenothiazines and a drug known to have marked adrenergic blocking activity—raises the threshold for sympathetic excitation at the hypothalamic level, where the sympathetic nervous system has its headquarters. This suggests a blocking action of chlorpromazine against the sympathetic neurohumoral transmitter substance. Although Hess⁹ first suggested the possible existence of a distinct central sympathetic transmitter substance, it remained for Vogt¹⁰ and Brodie and Shore¹¹ to demonstrate the presence of norepinephrine in the hypothalamus. Norepinephrine, a precursor of epinephrine, is readily blocked by chlorpromazine. If norepinephrine is established as the true transmitter substance necessary for excitation of the sympathetic center in the posterior hypothalamus, it may be reason-

ably assumed that chlorpromazine achieves its effect by preventing norepinephrine from exciting those nerve cells that generate widespread sympathetic effects. Confirmation for this postulate exists in the widespread reduction of sympathetic tone in the body by chlorpromazine. There is vasodilation and hypotension with compensatory tachycardia, relaxation of smooth muscle, and an alteration in pupillary size and salivary and gastric secretions. Most peripherally active adrenergic blocking agents fail to block the adrenergic influence on the heart.

Reserpine, as a representative of the rauwolfia group, acts in a somewhat different manner. It has the unusual property of causing a depletion in central stores of norepinephrine. Reserpine has been demonstrated to enter readily and leave the cells of the brain, but to exert a more lasting effect by interfering with the binding of norepinephrine in the cells. As quickly as norepinephrine is formed, it is dissipated. It fails to enter into preformed stores—a step presumably necessary for the subsequent excitation of the sympathetic effector cells. Thus, as with chlorpromazine, the result is a failure to transmit the intended neurohumoral message to the effector cells of the central sympathetics. In both instances, the tone of the sympathetic division of the autonomies is diminished. Again, evidence for this is the marked hypotension that results. Simultaneously, the balance of autonomic activity is shifted in favor of the parasympathetic system; this results in the increase in gastrointestinal activity and gastric secretion, bradycardia, pupillary constriction, and salivation. Thus, although the mechanisms of action are different for the rauwolfia and phenothiazine groups, their over-all effect is to decrease sympathetic, adrenergic activity (and ergotropic responses) and to favor parasympathetic, non-adrenergic (or trophotropic) activity. In either case, psychomotor agitation is lessened. This concept may not be correct; other modes of action now under investigation may in reality be occurring. However, the general concept exists of an action that raises the threshold of excitability of the sympathetics while simultaneously lowering the threshold of excitability of the parasympathetics.

The major tranquilizers are able to suppress central sympathetic activation without significantly depressing the reticular activating system; this favors the control of psychomotor agitation without significant loss of cortical activity or consciousness. Sleep does not occur. This is in marked contrast to the action of the barbiturates, which control the hyperactivity of the hypothala-

mus but also are prominently active in suppressing the reticular activating system. As a consequence of this latter action, sleep ensues and control of the emotional state occurs at the expense of loss in intellectual function.

Claims have been made for a true tranquilizing action of the substituted diols, but little evidence has been obtained for the kind of action exhibited by the major tranquilizers. Meproamate and phenaglycodel, as well as methaminodiazepoxide and chlormezanone, show striking activity on polysynaptic reflexes of the spinal cord, which suggests that their main mode of action is that of reducing muscle tension. Reduction of muscle tension decreases the number of afferent proprioceptive impulses which tend to aggravate existing tension and anxiety. This concept is confirmed by the fact that meproamate is useful in treating tension headaches. It has little effect in migraine headache and has little, if any, effect in the treatment of psychomotor agitation. In very high dosage, it simultaneously impairs motor and intellectual functions as do the barbiturates.

The mechanism of action of the diphenylmethanes is even more difficult to resolve. However, some evidence is available that use of benactyzine, for example, results in a significant block of conditioned responses. The compound has strong anti-cholinergic activity, but such activity, including the role of the autonomies in conditioned behavior, still needs to be elucidated.

PHENOTHIAZINE TRANQUILIZERS IN CURRENT USE

Since our classification of tranquilizers in 1958,⁴ a number of new drugs have been introduced—mostly of the phenothiazine type.

In general, all the phenothiazines are useful in relieving psychotic patients of emotional tension, agitation, and excitement. This emotional release may result in the fading of delusions and hallucinations and in the development of better personal integration and social behavior. Beneficial effects which are somewhat similar, but less clear-cut, may be obtained in withdrawn schizoid individuals and in many nonpsychotic patients.

This type of action is essentially similar with all effective phenothiazines. The individual compounds vary chiefly in their potency and in the nature and severity of their side reactions. These side reactions often determine the usefulness of any compound in the treatment of a given individual.

Synthetic developments of the phenothiazines have occurred along several lines: (1) in those

compounds closely resembling chlorpromazine with the dimethyl-amino-propyl side chain, (2) in those with a piperazine-propyl side chain, and (3) in those with a methyl-piperidyl side chain. Chemical differences of the subgroups and of the individual compounds may be recognized by referring to figure 1.

Dimethyl-amino-propyl phenothiazine subgroup

The dimethyl-amino-propyl subgroup is characterized by moderate potency with significant sedative action. Compounds in this group appear to be especially useful in psychomotor agitation and in manic, excited, and delirious states where the sedative properties of the drugs are desirable. Postural hypotension and skin eruptions are apt to occur with all members of this group. Blood dyscrasias, although rare, do occur.

Chlorpromazine (Thorazine), of course, is the most widely used of this group. It was the first phenothiazine to be introduced and we know most about it. It has a wide spectrum of useful activity but also produces all of the side reactions mentioned.

Triflupromazine (Vesprin) is at least twice as active as chlorpromazine, but otherwise it is similar in its spectrum of action.

Methoxypropazine (Tentone) is the most recent addition to this group. It is claimed that its capacity for producing disturbing side effects is minimal. Like promazine, it is weaker than chlorpromazine.

Promazine (Sparine) is perhaps the weakest of this group. It is less active than chlorpromazine but is a compound which exhibits very low parkinsonism. These less potent compounds may be less troublesome for ambulatory patients.

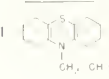
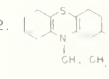
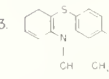
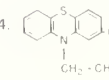
Piperazine-propyl phenothiazine subgroup

The piperazine-propyl subgroup has extremely high activity on a milligram basis. Drugs of this group are useful when less sedation is desired. Since they often are able to activate chronic withdrawn schizophrenic patients, they appear to have a type of stimulating action. These compounds have very high antiemetic activity. There is virtual freedom from certain side effects, such as postural hypotension and skin disorders (including solar erythema). These compounds produce little, if any, jaundice. The incidence of blood dyscrasias appears to be a good deal lower than with other phenothiazines; in fact, they may not occur at all.

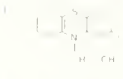
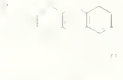



There is one main drawback to this group: extrapyramidal reactions, which roughly parallel efficacy on a milligram basis, are common and can be severe. Pseudoparkinsonism, akathisia

A Phenothiazine Group

a Dimethyl Amino-Propyl Subgroup

STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1. 	1-(3-dimethylamino-1-propyl)-2-methyl-10-phenothiazine	Promazine	Sparine
2. 	1-(3-dimethylamino-1-propyl)-5-chloro-2-methyl-10-phenothiazine	Chlorpromazine	Thorazine
3. 	1-(3-dimethylamino-1-propyl)-4-(trifluoromethyl)-2-methyl-10-phenothiazine	Triflupromazine	Vesprin
4. 	1-(3-dimethylamino-1-propyl)-10-methoxy-2-methyl-10-phenothiazine	Methoxypropazine	Tentone

b Piperazine Propyl Subgroup

STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1. 	1-(4-chlorophenyl)-4-[2-methyl-10-(1-methyl-4-piperazinyl)phenothiazinyl]-1-piperazine	Perchlorazine	Compazine
2. 	1-(4-methylphenyl)-4-[2-methyl-10-(1-methyl-4-piperazinyl)phenothiazinyl]-1-piperazine	Thiopropazine	Telazine
3. 	1-(4-chlorophenyl)-4-[10-phenothiazinyl]-1-piperazine	Perphenazine	Trilafon
4. 	1-(4-chlorophenyl)-4-[10-phenothiazinyl]-1-piperazine	Prochlorperazine	Prolixin, Permitil
5. 	1-(4-methylphenyl)-4-[10-phenothiazinyl]-1-piperazine	Thioridazine	Daridol

c Methyl-Piperidyl Subgroup

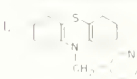
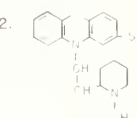
STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1. 	1-(1-methyl-3-piperidyl)-2-methyl-10-phenothiazine	Mepazine	Pacatal
2. 	Methyl-4-(4-methylphenyl)-1-piperazine-1-yl-10-phenothiazine	Thioridazine	Mellaril

Fig. 1. Tranquilizing and related drugs: listing of phenothiazine group. (a) Dimethyl-amino propyl subgroup, (b) piperazine-propyl subgroup, and (c) methyl-piperidyl subgroup.

(motor restlessness), dystonic symptoms, and sensations of weakness of the extremities appear more readily with this group than with other phenothiazine drugs. The experienced clinician will have no difficulty in recognizing these reactions; they may be controlled by dosage reduction or the use of antiparkinsonian drugs, such as benztropine methanesulfonate (Cogen-

tin). Since susceptibility to extrapyramidal reactions varies greatly, some physicians routinely administer antiparkinsonian drugs from the time treatment with large doses of a piperazine-type compound is initiated until the individual's susceptibility has been determined. Although these reactions are not serious, they can be unpleasant and frightening for the patient.

Prochlorperazine (Compazine) is an agent that shares high potency—approximately 4 times that of chlorpromazine—and rapid effect with other members of this group. Although promoted for neurotic and psychosomatic individuals, Hippus and Kanig¹² first pointed out that this drug is useful in the treatment of catatonic and retarded individuals.

Trifluoperazine (Stelazine) is fast-acting and is approximately 10 times as active as chlorpromazine. Because of its notable stimulating action, it has recently been found useful in treating psychiatric depressions.

Perphenazine (Trilafon) is also a potent compound with a wide spectrum of useful activity. It is approximately 6 times as potent as chlorpromazine on a milligram basis.

Fluphenazine (Permitil, Prolixin) is, in terms of milligram potency, the most active compound available at the present time. It has an interesting chemical relationship to both trifluoperazine and perphenazine. It is considered to be more than 20 times as potent as chlorpromazine.

Thiopropazate (Dartal) shares with prochlorperazine its relative therapeutic effectiveness and produces somewhat similar side effects.

Methyl-piperidyl phenothiazine subgroup

The 2 members of this subgroup are not much alike, but they do have certain chemical features in common. In addition to this, the incidence of parkinsonism from these drugs is very low and their milligram potency is somewhat less than that of chlorpromazine.

Mepazine (Pacatal) has been noted to have a slight euphorogenic effect, but its use is associated with rather severe atropine-like side effects. Impairment of bladder function commonly occurs. In well-controlled studies, the compound has not appeared too effective in treating chronic psychotic disorders.

Thioridazine (Mellaril) has a broad spectrum of useful activity. It is considered by many to be a good all-round phenothiazine tranquilizer. The compound has virtually no antiemetic activity. It produces relatively few side effects, although postural hypotension is associated occasionally with its use and reversible toxic retinitis has occurred with excessive doses.

SIDE EFFECTS WITH THE PHENOTHIAZINES

Phenothiazine drugs have a high incidence of side effects and complications. Individuals vary tremendously in their ability to tolerate these compounds. Some patients are not troubled by any of them and others have great difficulty with almost all of them. From the practical point of view, difficulty with side reactions may determine the effectiveness of the compound, particularly in ambulatory patients. Finding a drug that a given patient can tolerate may involve a trial and error search, but the following general principles may serve as a rough guide.

Common side reactions which may occur with any of the phenothiazines include dry mouth, blurred vision, constipation, unwanted weight gain, edema, vivid dreams, and seizures (usually with very high doses only). Any of these reactions may occur in the susceptible individual and they usually cannot be avoided by switching to another phenothiazine at the equivalent dosage level. When they occur, they must either be endured or controlled through reducing the dosage or instituting other measures. As an exception, mepazine has a marked tendency to produce atropine-like symptoms; if dry mouth, blurred vision, and urinary disturbances occur with its use, relief may be obtained by shifting to another compound.

Other side effects that may be lessened or avoided by shifting from one drug to another include extrapyramidal phenomena, postural hypotension, skin disturbances, jaundice, and leukopenia.

As mentioned previously, extrapyramidal phenomena are most likely to occur with a piperazine-type compound. However, there is marked variation in individual susceptibility. Some patients can take large doses of a piperazine compound without any difficulty developing. In others, a reaction may develop rapidly and dramatically on a very small dose. If a patient proves highly sensitive to these neurologic side reactions, it may be advisable to prescribe one of the compounds which has a low tendency to produce this type of disturbance. These compounds are thioridazine, promazine, mepazine, and methoxypromazine. Relative freedom from this extrapyramidal type of disturbance is also claimed for the newly introduced compounds, prothipendyl and chlorprothixene.

Dermatitis, jaundice, leukopenia, and postural hypotension are 4 side reactions which apparently occur very rarely, if at all, among the piperazine-type compounds. Leukopenia is the most serious complication that is encountered with the phenothiazine drugs. It is not always

possible to know the cause of the leukopenia; but most of the phenothiazine compounds, with the possible exception of the piperazines, have been implicated.

Jaundice of an obstructive type has occurred principally with chlorpromazine and seldom with any of the other compounds. Fortunately, this is a reversible hepatic disorder that subsides after cessation of the drug.

Dermatologic eruptions are usually quite mild and can be controlled by discontinuing the drug. It is often possible to restart the same drug later without recurrence. On the other hand, it may be more practical to shift to one of the piperazine compounds, since these usually do not produce skin reactions.

Postural hypotension is usually mild with compensatory adjustment occurring in a few days. However, it can be serious for highly susceptible persons or for the elderly in whom falls may result with possible injury.

The piperazine compounds are relatively free of the most disturbing side reactions, except for those of the extrapyramidal type. Many physicians believe that these neurologic phenomena may be related to the therapeutic effectiveness of neurotropic compounds. A relative absence of the sensitivity type of side reaction in the piperazine compounds may be partly related to the fact that, because of their greater potency, the dose is low. This is one reason for attempts by the pharmacologist to find more potent compounds. The smaller the dose, the less likely that sensitization or unusual reactions will occur. The advantages of the piperazine subgroup illustrate this concept.

DOSAGE PROCEDURES FOR THE PHENOTHIAZINES

It is not possible to give arbitrary dosage procedures for most of the neuroleptic agents. Virtually all authors stress the fact that the dose needs to be adjusted individually, depending upon the severity of the patient's symptoms and his individual tolerance to a given compound. Since this tolerance is unknown initially, it is usually wise to begin with a modest dose and increase the medication progressively until the desired effect has been attained. In most physically healthy patients, this can be done quite rapidly. Older people are an exception—most of them cannot tolerate high doses without developing mental confusion, ataxia, postural hypotension, or other disturbing side reactions.

In treating ambulatory patients—especially those who drive cars or go to work—avoiding or controlling side effects is of paramount importance. For this reason, the physician should

stay within the low dosage levels recommended by the manufacturer for ambulatory patients.

In treating more severely distressed individuals or those who are delirious, fairly high doses or possibly a parenteral route of administration may be needed. Since some patients are very tolerant to the phenothiazines, the question often arises as to whether failure to obtain clinical improvement results from insufficient dosage. The occurrence of drowsiness, tremors, ataxia, extrapyramidal phenomena, or mental confusion is suggestive evidence that brain functions are being altered by the medication. When, in the face of these signs of central nervous system effect, clinical improvement fails to accrue after a period of time, the clinician may feel assured that failure of treatment is not due to inadequate dosage.

After attaining a favorable clinical response, medication is usually reduced to a maintenance dose. Ideally, this is a dose which will continue to provide the patient with calming action but will be free of side reactions. If the patient has been very distressed, reduction to a maintenance dose should be carried out slowly and cautiously.

Present evidence indicates that some chronic mental patients need maintenance therapy indefinitely if they are to avoid relapse. Many such persons are being maintained on fairly high doses of chlorpromazine without apparent harm.

THE RAUWOLFIA GROUP OF TRANQUILIZERS

The rauwolfia alkaloids (figure 2) require minimal dosage and are relatively inexpensive. On the other hand, they are slow in onset of action, they produce gastric hypersecretion, and they have activated peptic ulcers. Even more serious,

B	Rauwolfia Group			
	STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1		Reserpine	Reserpine	Serpasil
2		Deserpidine	Deserpidine	Modurit
3		Deserpine (Reserpine)	Deserpine (Reserpine)	Harmoni

Fig. 2. Tranquilizing and related drugs: listing of rauwolfia group.

they have induced a relatively high incidence of psychiatric depressions. Physicians are cautioned in the use of these drugs because of an associated high incidence of suicides.

As pointed out previously, the over-all tranquilizing effects of the rauwolfia alkaloids are similar to chlorpromazine, although the drugs act through different mechanisms. Chlorpromazine appears to block ergotropic or sympathetic functions. Reserpine, on the other hand, stimulates the trophotropic system. Ergotropic activity is associated with alertness, energy, and activity. In contrast, trophotropic action is associated with rest, economy, repair, and general welfare of the organism.

Reserpine, of which there are many brand names and forms, is the parent drug of this series. It is used in the treatment of excitatory and manic states as well as hypertension.

Rescinnamine (Moderil), chemically similar to reserpine, produces many of the same actions. Like reserpine, it should be used with caution in patients with a history of peptic ulcer or depressive tendencies.

Recanescine or deserpidine (Harmony) also has many of the actions of reserpine but is claimed to be relatively free from many of the side effects of the rauwolfias. It should be used cautiously in peptic ulcer, in epilepsy, and in patients about to undergo surgery or electroshock therapy.

POSSIBLE INTERMEDIATE RANGE TRANQUILIZERS

The search for more effective and less toxic compounds results from time to time in the appearance of new drugs. The following 2 compounds are chemically related to the phenothiazines. They are too new to allow definite classification, but early evidence indicates that they might be considered intermediate range tranquilizers (figure 3).

Prothipendyl (Timovan) appears to have antipsychotic potency approximating that of promazine. It has had extensive use in Europe, but has only recently been introduced into the United States. Evidence indicates that it has very few side effects; postural hypotension is the most significant. So far it has not produced extrapyramidal symptoms even at high dosage. Since it does not meet all the criteria that we have set forth for the major tranquilizers, we have felt it wise to include it for the moment, at least, among the intermediate range group.

Chlorprothixene (Taractan) is a derivative of thioxanthene, but otherwise it is structurally related to chlorpromazine. This new compound

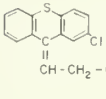
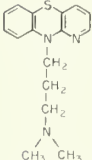
STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1 	2-chloro-9-(3-dimethyl-aminopropylidene)-thioxanthene	Chlorprothixene	Taractan
2. 	4-dimethyl-4-(3,4-dihydro-1,4-benzothiazin-2-yl)-1,4-benzothiazine	Prothipendyl	Timovan

Fig. 3. Tranquilizing and related drugs: listing of possible intermediate range tranquilizers.

evidently has some antipsychotic activity. It has a number of side effects similar to those of the phenothiazine drugs, but these are relatively mild. As with prothipendyl, this compound is claimed to have antidepressive action somewhat similar to that of imipramine. Evidence indicates that it does not produce the parkinsonism or other extrapyramidal phenomena characteristic of the major tranquilizers.

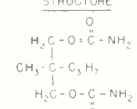
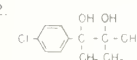
MINOR TRANQUILIZERS

The minor tranquilizers (figure 4) are very widely used and are the preferred drugs in common nervous illnesses. Because of their relative safety, many physicians prefer to try them first in any ambulatory case. In many instances, their calming action is sufficient to help the patient attain relief during the acute phase of his nervous disorder. They may be particularly useful in mild depressions and in many psychosomatic disturbances. Many of these illnesses are of a self-limiting nature; it is usually not necessary for the patient to continue use of minor tranquilizers over any extended period of time. Many patients find them especially helpful during periods of stress.

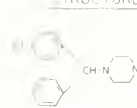
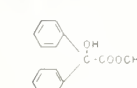
In spite of the wide use and great popularity of the minor tranquilizers, much less is known about them than is known about the majors. It is more difficult to evaluate and run controlled studies on less severely ill patients. Changes brought about by use of these drugs are less marked and therefore more difficult to measure and evaluate. In spite of this lack of objective and scientific evidence, the great popularity of the minor tranquilizing group does support the contention that they are of value in the relief of many nervous symptoms.

1. *Substituted diol group.* Meprobamate (Miltown, Equanil) and phenaglycodol (Ultran) have a mild sedative action with muscle-relax-

A Substituted Diol Group

STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1. 	2-methyl 2-n-propyl 1,3-propanediol dicarbamate	Meprobamate	Miltown Equanil
2. 	2-(p-chlorophenyl) 3-methyl-2,3-butanediol	Phenaglycodel	Ullian

B Diphenylmethane Group

STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1. 	1-(p-chlorobenzhydryl)-4-[2-(2-hydroxyethoxy)-1-ethyl]piperazine	Hydroxyzine	Atarax Vistaril
2. 	2-dimethylaminoethyl benzoate	Benactyzine	Suavital

C Miscellaneous Group

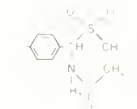
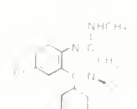
STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1. 	1-(2-methyl-5-phenyl-1,3,4-oxadiazol-5-yl) ethanone	Chlormezanone	Trancopal
2. 	7-methyl-5-phenyl-1,3-diazepine-2,6-dione	Methaminodiazepoxide	Librium

Fig. 4. Tranquilizing and related drugs: listing of minor tranquilizers. (a) Substituted diol group, (b) diphenylmethane group, and (c) miscellaneous group.

ing and some anticonvulsive activity. Meprobamate is the most widely used of all tranquilizers. Although it is a safe compound compared with the major drugs, habituation, ataxia, and skin reactions occur occasionally.

2. *Diphenylmethane group.* Hydroxyzine (Atarax, Vistaril) and benactyzine (Suavital) are antonomic suppressants that are capable of blocking certain conditioned reflexes. Side effects are minimal. These drugs have been useful in treating many patients with nervous tension. Because there is little or no tendency to habituation, they have been successfully used with alcoholics.

3. *Miscellaneous group.* Methaminodiazepoxide (Librium) is a newly introduced and potent central muscle relaxant which has sedative and tranquilizing action. It is close to meprobamate but is much more potent and broader in its profile of action. Some investigators consider it an intermediate range tranquilizer. Chlormezanone

(Trancopal) is another central muscle relaxant with mild tranquilizing activity.

MISCELLANEOUS PSYCHOTHERAPEUTIC DRUGS

Bucizine (Softran), phenyltoloxamine (P.R.N.), captodiamine (Suvren), and promoxolane (Dimethylane) have not been evaluated sufficiently to determine whether or not they have a place among the effective psychotherapeutic agents.

Amphenidone (Dornwal) is a newly introduced compound that may be of value in the treatment of nonpsychotic disorders. It is a central muscle relaxant with mild tranquilizing effects and apparently no sedative action (figure 5).

ANTIDEPRESSANTS

The antidepressants are drugs useful in the treatment of psychiatric depressions. Most drugs in this class act by stimulation; they increase alertness of the central nervous system and elevate the mood of the depressed patient. As will be pointed out later, some presumably act by other mechanisms. Because none of the mechanisms completely describes the actions of all compounds, it is believed that there are additional actions not yet known. In general, however, most compounds of the stimulating class can be classified either as direct or indirect stimulants. Some drugs, by virtue of a combination of both actions, can be considered bimodal stimulants. Reference to figure 6 originally presented by Schiele and Benson,¹³ diagrammatically aids in understanding the concept of a bimodal stimulant.

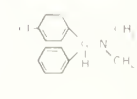
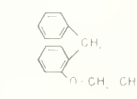
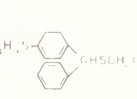
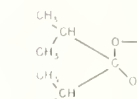
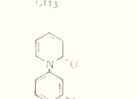
STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
1. 	1-(2-methyl-5-phenyl-1,3,4-oxadiazol-5-yl) ethanone	Bucizine	Softran
2. 	1-(2-methyl-5-phenyl-1,3,4-oxadiazol-5-yl) ethanone	Phenyltoloxamine	P.R.N.
3. 	1-(2-methyl-5-phenyl-1,3,4-oxadiazol-5-yl) ethanone	Captodiamine	Suvren
4. 	1-(2-methyl-5-phenyl-1,3,4-oxadiazol-5-yl) ethanone	Promoxolane	Dimethylane
5. 	1-(2-methyl-5-phenyl-1,3,4-oxadiazol-5-yl) ethanone	Amphenidone	Dornwal

Fig. 5. Listing of miscellaneous psychotherapeutic drugs.

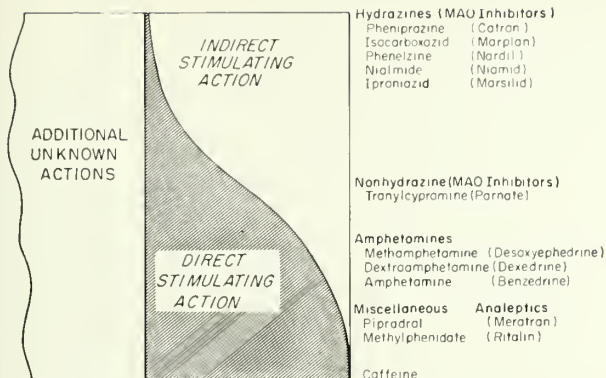


Fig. 6. Diagrammatic representation of various mechanisms of central nervous system stimulation.

POSSIBLE MODES OF ACTION OF THE ANTIDEPRESSANTS

Although the mechanism of action of the antidepressant drugs is largely unknown, a good deal of speculation has been offered. Some drugs such as the older analeptics and amphetamines, appear to have a direct excitatory effect on neuron cells; others, particularly the hydrazines, appear to act in an indirect manner to activate the nervous system. The possibility exists that some agents increase the available amount of neurohumoral transmitter substance, thus increasing central nervous system activity. The amine-oxidase inhibitors presumably act in this manner to increase the concentration of catecholamines and indoleamines. One other type of agent (deanol), used to increase central activity, is considered to be a precursor substance for acetylcholine. Theoretically, it can reinforce central cholinergic excitation.

Other agents in use have a beneficial effect in the clinical situation because of their suppressant properties. Some phenothiazines and sedative and hypnotic agents apparently act to relieve the agitation that contributes to certain types of depression. Besides, there may be agents, such as imipramine (Tofranil), which, while possessing suppressant properties, may also sensitize receptors to noradrenaline in much the same way as cocaine sensitizes receptors to the action of adrenaline. Central nervous system receptors for adrenaline or noradrenaline have not been demonstrated to exist. Nevertheless, the possible existence of central adrenergic receptors is acknowledged by the presence of synthesizing and metabolizing enzymes in the diencephalic areas of the brain and by the adrenergic substances themselves. One area in which activation could occur is the ergotropic

area schematically represented by the stippling in figure 7.

CLASSIFICATION AND DISCUSSION OF THE ANTIDEPRESSANTS

The recent interest in psychotherapeutic agents useful for alleviating depressions prompted the authors to classify the available drugs. Even though the classification remains tentative, it serves the function of guiding the practitioner in the selection of appropriate medications.

Central Nervous System Stimulants

Predominantly direct stimulants. Figure 8 lists the central nervous system stimulants that are characterized by predominantly direct action, as pointed out by Schiele and Benson.¹³ They have the following distinguishing characteristics:

1. They act to stimulate the nervous system directly, with little or no monoamine-oxidase inhibition.
2. Their action is rapid and brief; the effect wears off in a matter of hours and may be followed by a letdown.
3. They tend to have a hypertensive effect. The amphetamines, particularly, exhibit pressor activity and are regarded as moderate sympathomimetic agents.
4. They tend to reduce appetite and are widely used clinically to control obesity.
5. They are relatively nontoxic.
6. Habituation or dependency has been a problem with some of them.

The amphetamines—Benzedrine, Dexedrine,

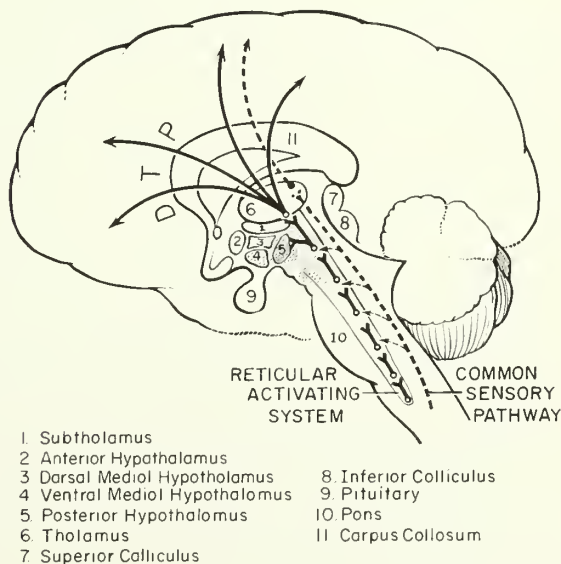
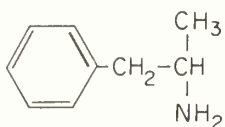


Fig. 7. Schematic illustration of mesial section of brain with neural connections.

A. PREDOMINANTLY DIRECT STIMULATION

STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
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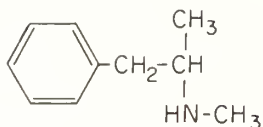
1. MODERATE SYMPATHOMIMETICS



a. d,l-1-Phenyl-2-aminopropane

Amphetamine

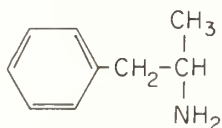
Benzedrine



b. d,l-1-Phenyl-2-methylaminopropane

Methamphetamine

*Desoxyephedrine**

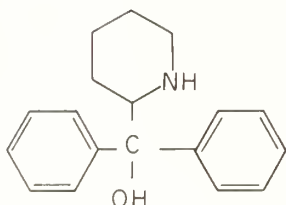


c. d,l-1-Phenyl-2-aminopropane

Dextroamphetamine

Dexedrine

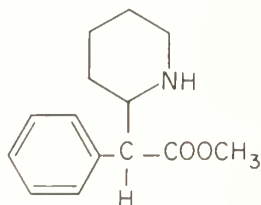
2. MINIMAL SYMPATHOMIMETICS



a. α -(2-Piperidyl) benzhydrol

Pipradrol

Meratran

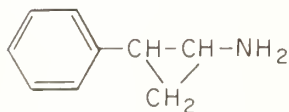


b. Methyl α -phenyl-2-piperidineacetate

Methylphenidate

Ritalin

B. BIMODAL STIMULATION



a. Trans-dl-2-phenylcycloproylamine

Tranylcypromine

Parnate

*One of several applicable trade names

Fig. 8. Listing of central nervous system stimulants.

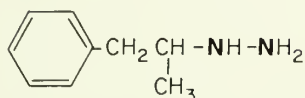
Desoxyephedrine, and so forth—are characterized by their moderate sympathomimetic action, their anorexic effect, and their mild euphorogenic activity. Unfortunately, their brief response is followed by a considerable rebound effect of post-treatment depression.

Pipradrol (Meratran) and methylphenidate (Ritalin) act somewhat similarly but exert less excitation and anorexia. Their stimulating action is associated with only moderate autonomic effects. The duration of their action is relatively short.

C. PREDOMINANTLY INDIRECT STIMULATION

STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
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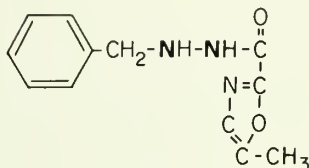
1. MONOAMINE-OXIDASE INHIBITORS (HYDRAZINE GROUP)



a. β -Phenylisopropylhydrazine

Pheniprazine

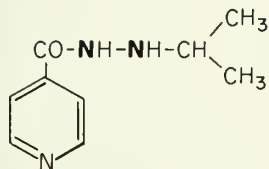
Catron



b. 1-Benzyl-2-(5-methyl-3-isoxazolylcarbonyl)hydrazine

Isocarboxazid

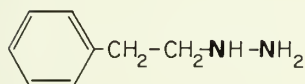
Marplan



c. 1-Isonicotinyl-2-isopropylhydrazine

Iproniazid

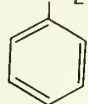
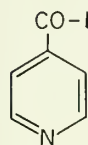
Marsilid



d. β -Phenylethylhydrazine

Phenelzine

Nardil

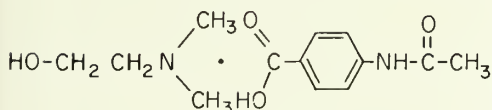


e. N-Isonicotinoyl-N'-[β -(N-benzylcarboxamido) ethyl]-hydrazine

Nialmide

Niamid

2. CHOLINERGIC REINFORCER



a. Paracetamidobenzoic acid salt of 2-dimethyl-aminoethanol

Deanol

Deaner

Fig. 9. Continued listing of central nervous system stimulants.

Bimodal stimulants. *Tranylcypromine* (Parnate) is a nonhydrazine derivative of amphetamine; it possesses monoamine-oxidase-enzyme inhibiting action. Because it also exerts direct stimulating activity, it is of special interest. It is considered a bimodal stimulant.

Predominantly indirect stimulants (figure 9). *Monoamine-oxidase inhibitors (hydrazine group)*: the distinguishing features of these drugs, as previously outlined by the authors, are the following:

1. Their stimulating action is believed to be mediated by their monoamine-oxidase (MAO) inhibiting action or by related phenomena. They

may have slight direct central nervous system stimulating activity.

2. Their stimulating action is slow in onset, often requiring days or weeks to become apparent. This action, however, is prolonged and tends to be cumulative, with no subsequent let-down.

3. These agents frequently give rise to postural hypotension in contrast to the pressor activity that is often observed with direct-acting stimulants.

4. Their use is accompanied by increased appetite and, commonly, by constipation.

5. They produce a relatively high incidence

of troublesome side reactions, partly because of their cumulative action. Serious and even fatal toxic disturbances have occurred with iproniazid.

6. Agents in this group do not appear to induce habituation or drug dependency.

Representing the group of drugs with an indirect mode of antidepressant action is *iproniazid* (Marsilid). This hydrazine type of compound, like its congeners, requires a long duration for its action to become manifest. However, once its action is established, it is more sustained than the amphetamines or the piperidines. Iproniazid, in contrast to the amphetamines, increases the appetite; its effect in elevation of mood is significantly greater.

Drugs of this group are able to inhibit the enzyme that is capable of oxidizing certain essential amines, including the catecholamines (epinephrine and norepinephrine) and such indoleamines as serotonin (5-hydroxytryptamine). Blocking of the enzyme should lead to an accumulation of amines. It has not been demonstrated that an accumulation of norepinephrine is the basis for the action of the hydrazines. Nevertheless, the possibility remains that some essential amine is increased within the central nervous system and that it activates the ergotropic centers. Some results with these drugs have compared very favorably with the effects produced by electroshock in psychiatric depressions, but their use is not free of danger. Iproniazid, in particular, is prone to produce liver injury. The effect produced is not unlike that of viral hepatitis and, as a matter of fact, the suggestion has been offered that the drug can evoke latent hepatitis. With high dosage, other side effects, such as severe hypotension and frank psychomotor hyperexcitability, have occurred. It is hoped, of course, that the newer members of this series—pheniprazine (Catron), isocarboxazid (Marplan), phenelzine (Nardil), and nialamide (Niamid)—will obviate some of the undesirable toxic effects. It is still too early to be assured. Severe liver damage with pheniprazine has recently been reported.¹¹

Another drug believed to act indirectly is deanol (Deaner). This agent, the paracetamido-benzoic acid salt of 2-dimethylaminoethanol (figure 9), is believed to be a precursor substance for acetylcholine. Thus, theoretically, it reinforces the central excitatory state.

Central nervous system suppressants

Imipramine (Tofranil) possesses sedative properties similar to those of the phenothiazines. There is considerable uncertainty over its proper classification (figure 10), but virtually all

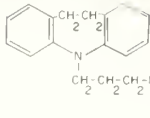
STRUCTURE	CHEMICAL NAME	GENERIC NAME	TRADE NAME
	1 5 (2-dimethylaminopropyl) 10,11-dihydro 5H dibenz (b,f)azepine hydrochloride	Imipramine	Tofranil
	2 Certain Major Tranquilizers		
	3 Various Minor Tranquilizers		
	4 Barbiturates and Other Sedative Drugs		

Fig. 10. Listing of central nervous system suppressants.

clinicians agree that this compound has a wide spectrum of useful activity. Patients with both mild and severe depressive disorders often respond favorably. Those with agitation usually require the addition of a phenothiazine, such as thioridazine. As with other neuroleptic compounds, it is often not possible, to predetermine whether a given patient will respond favorably to imipramine. In spite of causing a number of side reactions similar to those of the phenothiazines, such as postural hypotension, dry mouth, and constipation, it has a good safety record; severe or serious complications are rare or absent. Its chief disadvantage is that beneficial effects often do not appear until the drug has been administered several days or even several weeks. Perspiration is often induced by this compound and may be quite disturbing to some individuals.

Other sedative drugs may be said to have some antidepressant properties (figure 10). At least, they may relieve insomnia and anxiety. If they succeed, this contributes to the over-all relief of depression. Finally, a number of the newer phenothiazines, such as trifluoperazine (Stelazine), give the clinical appearance of stimulation in that retarded and regressed patients become more animated and active when taking them. These compounds appear to benefit many depressed patients, especially when their use is combined with one of the MAO inhibitors.¹⁵

SIDE EFFECTS, DOSAGE, AND INDICATIONS OF THE ANTIDEPRESSANTS

Table 2 presents, in a simplified version, the side effects, principal actions, and indications for the antidepressive drugs. The physician must always remember the marked individual variations in patients. Two people may have very similar symptoms and history, with 1 patient responding to a given therapeutic procedure and another failing to respond. The table lists the major side effects and gives some comparison between groups.

As with the tranquilizing agents, the dose for

TABLE 2
ANTIDEPRESSIVE DRUGS

Drug	Common side effects	Serious complications	Action and indications
DIRECT STIMULANTS			
<i>Moderate sympathomimetics</i>			
Amphetamine	Pressor effect	0	Rapid short-acting stimulation for depressed mood or drowsiness. Appetite control.
Methamphetamine	Overstimulation		
Dextroamphetamine	Insomnia		
	Habituation		
<i>Minimal sympathomimetics</i>			
Pipradrol	Overstimulation	0	Rapid short-acting stimulation for depressed mood or drowsiness.
Methylphenidate	Insomnia		
BIMODAL STIMULANTS			
Tranlycypromine	Insomnia Postural hypotension Headache Overstimulation	0	Rapid sustained stimulation nonagitated depressives ^o depressed schizophrenics ^o pseudoneurotic schizophrenics ^o depressed and fatigued neurotics ^o
INDIRECT STIMULANTS			
<i>Mao inhibitors</i>			
Pheniprazine	Postural hypotension	Liver toxicity	Slow, cumulative stimulation nonagitated depressives depressives refractory to other therapy. some schizophrenics ^o
Isocarboxazide	Constipation	Anemia	
Iproniazid	Tremors	Peripheral neuritis	
Phenelzine	Sweating	Psychotic disorganization in unstable	
Nialmide	Insomnia	personality (rare)	
	Overstimulation Edema		
<i>Cholinergic reinforcers</i>			
Deanol	Overstimulation	0	Mild stimulation nonpsychotic depressions.
CNS SUPPRESSANTS			
Imipramine	Postural hypotension Constipation Skin eruption Sweating	0	Slow, calming effect with improvement of mood. Any depressive disorder. Agitated depression requires major tranquilizer in addition.

*Usually combined with a Phenothiazine

antidepressive compounds must be individualized. Here too, there is marked variation in individual susceptibility and tolerance.

The monoamine-oxidase inhibitors, particularly the hydrazine compounds, are usually administered in a somewhat different manner from other neuroleptic drugs. Because of their slow onset, the beginning dose is usually high. Since these compounds are dangerous, the patient needs to be under regular medical supervision. The dose should be reduced to a maintenance level as soon as the patient begins to show a favorable response. The need to reduce dosage may be indicated by the appearance of such side reactions as insomnia, sweating, and tremor.

GENERAL DISCUSSION

From what has been stated, it is apparent that there are a number of contraindications to the

use of certain drugs. Because of their convulsant potentialities, the phenothiazines may not be tolerated by patients with low thresholds for seizures. In depressive states, the rauwolfia compounds are contraindicated because of their marked tendency to aggravate the condition. Conversely, in manic states, the antidepressant medications should not be administered because of their potentiality for increasing excitement. Among the latter drugs, the MAO inhibitors should be cautiously used or avoided completely in patients with evidence of hepatic disorders. And certainly for patients in whom blood dyscrasias are suspected, drug therapy of any type should be used advisedly.

The impression is rightfully gained that tranquilizing and antidepressant medications are associated with a high order of toxicity. Relatively speaking, this appears to be true. The risks

of therapy are fairly high. Nevertheless, for the first time in history, we have medications that provide significant therapeutic benefit to large numbers of psychiatric patients. As in the treatment of any disorder, the hazards of drug therapy must be weighed against the advantages. Surely, in time, medications will be improved just as they have been for other disorders. Greater specificity of action and lesser degrees of toxicity are goals to be achieved. Better diagnostic tools and physiologic guides also are needed. Ultimately, if and when inborn errors of metabolism are made known, genetic control of at least some disorders may be possible through alteration of the important genetic-bearing nucleic acids. If this can be accomplished, the physician may then have drugs which provide more than symptomatic control of these disorders. With persistent efforts, patients having nervous and mental illnesses—our most important medical and social problem—can be benefited much as those having bacterial infections were benefited by introduction of the sulfonamides.

SUMMARY

The recent rapid introduction of numerous psychotherapeutic agents necessitates organizing the drugs into chemical types with various actions and clinical indications. For the most part, the drugs consist of tranquilizers and antidepressants.

Most of the tranquilizers still fall into 2 groups: the majors—primarily useful in psychotic disorders, and the minors—useful in psychoneurotic illnesses and common nervous tension states. Both groups have their own subgroups.

Antidepressants are divided into the stimulants—direct and indirect—and the suppressants. The usefulness of the direct stimulants is limited by their short action. Indirect stimulants—the MAO inhibitors—provide sustained relief for many nonagitated, depressed psychotics. The suppressants are useful in agitated and anxious depressive reactions.

In general, these psychopharmacologic agents have facilitated the control of mood and behavioral disturbances. In the mental hospital setting, they have induced a more quiet and orderly atmosphere. They have enabled the discharge of an increasing number of patients and the return of many who were previously resistant to treatment to their appropriate places in society. In addition, they have facilitated ambulatory or outpatient treatment and have lessened the need for shock therapies.

Although many drugs are available and many are useful, they do have definite limitations. As a class, they possess a relatively high order of toxicity. Because these actions and toxicities vary considerably, it will be most satisfactory for the clinician to select 1 or 2 drugs from each group and become well-acquainted with these. If this is done, the drugs can aid the physician in treating many patients who have not previously been adequately controlled. For optimum results, the use of psychotherapeutic drugs should be combined with other forms of treatment, including psychotherapy and group or occupational therapy.

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The authors wish to express their appreciation to *Postgraduate Medicine* for permission to reproduce a number of tables and figures.

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Infections of the Nervous System

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INFECTIONS of the nervous system are no doubt much more frequent than generally suspected. Many of the milder infections are completely overlooked because of the paucity of dramatic neurologic manifestations or because of a failure to carry out an examination of the nervous system. Generally, nervous system infections may implicate the meninges, producing a meningitis, or the parenchyma, resulting in encephalitis or myelitis. However, rarely does either type of involvement occur independently, and, in most cases, both are implicated to result in meningoencephalitis.

Central nervous system infections may be caused by a wide variety of infective agents, ranging from virus to large animal parasites, such as ascaris and the tapeworms. Between these two extremes are the rickettsiae, the bacteria, and the spirochetes. These diseases are transmitted to man by practically every known method. Many, such as brucellosis, yellow fever, and tularemia, are maintained in animal reservoirs and periodically infect man through contact, contaminated food, water, or insect vectors. Some, such as typhoid fever and amebiasis, are transmitted directly from one individual to another by droplet infection or through contaminated food, water, or flies. Many of the tropical diseases, such as malaria, filariasis, and schistosomiasis, before attacking man must pass through a cyclic development in insect vectors or in aquatic hosts, such as fish or snails. Because many types of infective agents can cause nervous system infections, only the more common ones will be discussed, and these will be classified according to the nature of the infective agent.

GENERAL CLINICAL MANIFESTATIONS

The clinical features of meningoencephalitis are so diverse and numerous that a mere recital of the many variations alone would be a formid-

able task. The symptomatology varies from case to case, from epidemic to epidemic, and from one specific type of infection to another. There is so much overlapping in the symptomatology that it is not possible to identify a specific type of cerebral infection from the cerebral manifestations alone. However, certain features may predominate in specific types of infections, and these will be emphasized.

The presenting symptoms may consist of those characteristic of the general infection, such as malaise, lassitude, pains in the neck and back, sore throat, and mild fever with diffuse headache lasting a few days to a week. When gastrointestinal symptoms predominate, the patient may complain of vomiting, diarrhea, and abdominal discomfort. Very often, the neurologic symptoms are minimal and consist only of mild headache, slightly elevated temperature, and a mild *neural rigidity*. Transient focal complaints of *diplopia* or *vertigo* may vaguely suggest the possibility of a nervous system infection and result in the performance of a spinal tap. The latter will reveal a pleocytosis, chiefly of mononuclear cells.

In many cases, the persistence and intensity of the *headache* and the associated lassitude that tends toward a definite *drowsiness* suggest the possibility of meningoencephalitis. In children, early in the illness definite irritability, restlessness, or even convulsions may develop.

The usual well-recognized clinical form of nervous system infection is characterized by (1) signs and symptoms of a mild systemic involvement, (2) evidence of central nervous system involvement, and (3) manifestations of a rather diffuse or widespread implication of the nervous system.

Most frequently, the illness is ushered in abruptly with headache, moderately elevated temperature, chills, lassitude, and myalgia. As the illness progresses, the evidence of nervous system involvement becomes more apparent. The temperature remains elevated, and the headaches persist or become more intense. Stiff neck may

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be present, occasionally preceded by stiffness of the spine and pain in the limbs. Restlessness develops within a few days and is associated with or followed by lassitude and drowsiness. The intensity of the manifestations of the various symptoms varies from patient to patient.

The fever is variable and may be minimal. Typically, it is constant and low grade, often being highest during the first week of illness. In a few cases, the fever may rise to very high levels and may persist for long periods, depending upon the nature of the illness.

Mental symptoms are very common and show a wide range of manifestations. In adults, there is a tendency toward lassitude, apathy, drowsiness, and, finally, complete or partial unresponsiveness. In children, a period of irritability and restlessness most often occurs, which is followed only after a few days by drowsiness and even coma.

The physical findings are most variable, both in nature and in degree. Gross focal abnormalities usually do not occur. Pathologic reflexes may be encountered and often fluctuate from day to day. The deep reflexes show every possible variation and may be unequal, depressed, or exaggerated. In children, muscular rigidity, involuntary movements, or mild transient muscle weakness or paralysis often occurs.

The most diagnostic feature is the spinal fluid findings. The fluid is normally clear and under normal or slightly increased pressure. There is usually a moderate lymphocytic pleocytosis, ranging from 20 to 100 cells but usually not more than 50. In very acute cases, some polymorphonuclear leukocytes may be present. The spinal fluid protein is normal but may be slightly increased.

The course of the illness may be relatively brief, with apparent recovery in two weeks. In the average case, depending upon the etiology, the symptoms continue for about two weeks before a slow convalescence ensues. Exacerbations may occur, manifested by a transient return of fever, headache, and drowsiness.

Occasionally the illness may be fulminating in nature with an abrupt onset of headache, vertigo, and very high fever. Within a few hours, the patient has a stiff neck and some disorientation with restlessness and passes into coma. In children with this form of the illness, convulsions, motor weakness, and involuntary movements may develop. Death occurs in two to four days, either from bulbar symptoms or cardiac failure. If the patient survives the illness, the symptoms tend to subside fairly rapidly, but convalescence is prolonged by the persistence of

vague complaints of dull headache, vertigo, and lassitude for many weeks or even months.

SPECIFIC TYPES OF INFECTIONS

Viruses

Poliomyelitis. This is probably the most common viral disease of the nervous system. The disease spreads through oropharyngeal and fecal contaminants. The virus apparently multiplies in the lymphatic tissues of the gastrointestinal tract, followed by a hematogenous dissemination to the nervous system.

Poliomyelitis occurs most frequently in the late summer and fall and is often introduced by upper respiratory or gastrointestinal symptoms, such as abdominal pain, vomiting, or diarrhea. These symptoms are often accompanied or followed by evidence of meningeal involvement consisting of headache, backache, marked tightness of the back and neck muscles, and a positive Kernig's sign. The temperature is mildly elevated and the patient may be irritable or lethargic. No focal neurologic findings appear at this phase of the illness, but scattered deep reflexes may be suppressed. A spinal tap will reveal a mild pleocytosis up to 100 cells.

At this stage, the illness presents a picture of lymphocytic meningitis rather than encephalitis. The entire illness may become arrested at this stage, with complete recovery in a week. Such a case is referred to as the abortive form of the disease.

In some cases, the illness progresses, resulting in scattered motor weakness or bulbar palsy, with particular difficulty in swallowing. Such cases often show muscle tenderness, especially in the neck, back, and thighs. The prognosis now depends upon the severity of the motor involvement. Crippling occurs in about 20 per cent of clinically recognizable cases.

The diagnosis of poliomyelitis should always be suspected in a case of lymphocytic meningitis occurring in late summer and fall, particularly in the presence of tight back and neck muscles and scattered, decreased deep reflexes or focal muscle weakness.

Treatment depends upon the severity of the illness. In the abortive form, only symptomatic therapy is indicated. Muscle weakness requires special measures and exercises to prevent deformities. Respiratory insufficiency due to bulbar involvement requires careful attention to the airway, which must be kept clear by postural drainage, suction, or even tracheotomy.

Mumps. The frequency and severity of this nervous system involvement varies considerably from epidemic to epidemic. The mild cases are

often overlooked, and only the more severe forms of meningoencephalitis are recognized. The nervous system complications usually occur from the third to the fourteenth day after the onset of the parotid swelling but may precede the parotitis by days, while occasionally no parotitis occurs.

With mumps, either the meningeal or the cerebral symptoms may predominate. Onset of the illness is sudden, with intense headache, moderate fever, stiff neck, and mild somnolence. These symptoms may persist for seven to ten days and rapidly clear up. The spinal fluid usually contains less than 50 mononuclear cells and presents a picture of lymphocytic meningitis.

In some cases, the symptoms are rapidly progressive. The drowsiness becomes profound and occasionally is associated with convulsions. Motor weakness of an upper motor neuron type may be present, and the deep reflexes are often unequal and hyperactive. This cerebral form of mumps also tends to subside fairly rapidly after seven to ten days, with complete recovery in spite of the apparent gravity of the illness.

The spinal cord may be predominantly involved, resulting in severe motor and sensory disturbances. The prognosis in this form of the illness is grave and usually leaves considerable residual effects due to persistent cord damage.

The diagnosis should be suspected in any patient with high fever, severe meningeal or cerebral symptoms, and a lymphocytic meningitis who has a history of mumps or exposure to mumps or who develops an orchitis or pancreatitis. The diagnosis can be verified by the antihemagglutinin test.

Arthropod-borne virus infections. The two most prevalent of these viruses in this area are the western equine and the St. Louis viruses. Although both types have occurred in large epidemics, they are most commonly encountered as a sporadic illness. The disease appears only in the summer months and disappears when the weather turns cold. The vector for both these viruses is the mosquito. The actual reservoir is not known, although birds have been suspected.

The clinical features of these virus infections are not distinctive. Onset is usually sudden, with malaise, headache, and a low grade fever. The headache increases in intensity and is followed by drowsiness, which deepens to lethargy; however, the patient usually responds to sufficiently intense stimuli. Photophobia and vertigo may form a prominent part of the syndrome. Meningeal signs are usually mild but may be absent. A severe muscular weakness may be present and often persists long after all other disturbances

have subsided. Occasionally, all the deep reflexes are suppressed.

The course is usually favorable. Recovery is rapid and complete. About 10 per cent of the cases are fulminating and end fatally in seven to ten days. Children under 1 year of age often develop severe residual effects consisting of mental retardation, seizures, and behavior abnormalities.

The diagnosis of these viral encephalitides can be established by serologic tests. Specimens of the patient's serum are collected at about two-to three-week intervals, the first specimen being taken as soon after onset of the illness as possible. An increase in the antibody titer in the two specimens is usually diagnostic of the illness.

Enteric viral infections. A number of viruses which have a tendency to produce infections of the nervous system have been isolated from the stool. The two best known are the Coxsackie and the Echo viruses. Generally, they produce meningeal involvement with headache, stiff neck, and some malaise. The Coxsackie virus may cause marked muscle weakness with normal reflexes and, occasionally, chest pain due to a pleurodynia. Certain Echo strains produce a typical skin rash in addition to the meningeal involvement.

The spinal fluid reveals a mild increase in lymphocytes, usually under 50. The prognosis is generally good, with complete recovery in two to four weeks.

The diagnosis can be established most easily by the isolation of the virus from the stools.

Infectious mononucleosis. The exact etiology of this disease is not known, but it is believed to be of viral origin. The disease chiefly affects young adults. Although the clinical features are variable, the diagnosis can usually be made from a careful evaluation of the presenting symptoms. The illness often presents with a fluctuating low grade fever accompanied by sore throat, lymphadenopathy, splenomegaly, leukopenia with lymphocytosis, and, occasionally, jaundice. The most frequent neurologic symptom is headache, often associated with mild meningitis or encephalitis. The spinal fluid shows a lymphocytic pleocytosis and a mildly elevated protein value. Drowsiness, reflex alterations, and even papilledema have been observed.

Diagnosis usually can be made from the clinical symptoms and can be verified by the presence of a positive heterophil titer about two weeks after onset of illness. Treatment is symptomatic.

Bacteria

Meningococcus. This is a gram-negative diplococcus that primarily produces a purulent menin-

gitis. The disease is spread by droplet infection from the upper respiratory passages of infected individuals.

The clinical picture is typical of that of a purulent meningitis. Onset is abrupt, with rapid progression of symptoms. Headache occurs early and increases in intensity. Temperature becomes elevated and is associated with chills. Convulsions are common in children. Delirium, followed by lethargy or even stupor, may appear in the severe cases. Petechial hemorrhages may appear in the skin over the body. The most characteristic features are intense cervical pain, stiff neck on anteflexion, and positive Brudzinski's and Kernig's signs. The spinal fluid contains a greatly increased number of polymorphonuclear leukocytes, an increase in protein, and a decrease in sugar.

Diagnosis can be established by staining smears of the spinal fluid for the causative organism. Cultures of the blood and spinal fluid may also be helpful.

Sulfadiazine is specific for this infection. It may be combined with penicillin.

Pneumococcus, *Streptococcus*, and *Staphylococcus*. These organisms infect the nervous system by direct extension from an otitis media, a mastoiditis, or a suppuration in the accessory nasal sinuses or by a hematogenous route from an intrathoracic suppuration or a pyemia. They may localize within the meninges or the brain.

When the meninges are involved, a typical picture of a purulent meningitis results. The patients are very ill, with high temperature and very stiff neck. In most cases, there is some involvement of the underlying cerebral cortex, resulting in marked confusion, lethargy, and, often, focal neurologic disturbances, such as motor weakness or reflex abnormalities. The specific diagnosis can be established by spinal fluid smears or by blood or spinal fluid cultures.

Before treatment is instituted, the specific organism should be isolated and tested for sensitivity to the various antibiotics. The specific antibiotic should be administered in extremely high doses over a long period of time. Intramuscular or intravenous therapy is the best method of drug administration in order to counteract the cortical involvement.

Involvement of the brain results in a suppurative encephalitis that soon localizes to produce a brain abscess. These abscesses vary in size and location but frequently occur within the cerebellum or temporal lobes. The clinical picture is usually quite distinctive. Onset is often abrupt but may be slow and progressive. There are

definite signs of sepsis, with intermittent chills, malaise, fever, and leukocytosis. Focal signs soon appear, depending upon the location of the abscess. These focal findings are associated with evidence of increased intracranial pressure. The spinal fluid contains a mild pleocytosis, often with polymorphonuclear leukocytes predominating.

The diagnosis is generally readily suspected in any patient in whom signs of a space-occupying lesion in the setting of a generalized sepsis develop.

Treatment consists of the administration of specific antibiotics, depending upon the invading organism. This is followed by surgical emptying of the abscess.

Influenza. The organism responsible for influenza produces a purulent meningitis in children between 6 months and 2 years of age. Smears of the spinal fluid will reveal gram-negative encapsulated rods, which may be grown out on cultures of the blood or spinal fluid.

Treatment consists of a combination of chloramphenicol and sulfadiazine. If treated early, the prognosis is good. A number of patients develop subdural effusions, which result in persistent or recurrent cerebral symptoms after recovery. Subdural taps should, therefore, be performed in all cases in which the temperature is not normal within seven days after treatment is begun.

Tuberculosis. Tuberculous infections of the nervous system arise from some tuberculous focus within the body, usually the lungs or the tracheobronchial lymph nodes, with hematogenous dissemination to the central nervous system. The lesions remain intracerebral, resulting in a tuberculoma, or invade the meninges, resulting in tuberculous meningitis.

In children, this type of meningitis often presents with a very acute onset, which may be preceded by a period of malaise, weight loss, and irritability. The temperature may rise to high levels, the neck becomes stiff, and lethargy ensues. As the illness progresses, signs of increased intracranial pressure (vomiting and papilledema) and cranial nerve involvement may appear.

In adults, the course of the illness may be much more prolonged, with symptoms of malaise, weight loss, and low grade fever continuing for many months. At onset, headaches are mild and signs of meningeal irritation may be entirely lacking. As the illness progresses, headaches become more intense and isolated cranial nerve disturbances, chiefly diplopia, may appear.

The spinal fluid is usually sufficiently charac-

teristic to establish the diagnosis. The fluid is often clear but, on standing, shows a delicate weblike clot. The fluid contains a lymphocytic pleocytosis, an increase in protein, and a decrease in spinal fluid sugar to below 20 mg. per cent. On smear, acid-fast rods may be found in the spinal fluid. If organisms cannot be found, guinea pig inoculations should be done. The patient should be examined carefully for the original focus.

Treatment for tuberculous meningitis has considerably improved the prognosis of this very serious illness. The therapy of choice consists of a combination of intramuscular streptomycin and isoniazid.

Undulant fever (brucellosis). This infectious disease of animals is transmitted to man either through contact with the infected animal or by ingestion of their products, particularly infected milk.

Early in the illness, the patient develops headache, irritability, sleep disturbances, and retroorbital pain. In addition, there may be anorexia with weight loss, some night sweats, and periodic joint pain. As the disease progresses, there may be involvement of the nervous system, with vertigo, tremor, ataxia, lethargy, and intense headache. In some cases, the spinal cord is predominantly involved, resulting in segmental hypesthesia, radicular pain, motor disturbances, and sphincter involvement. Involvement of the lumbosacral spine may result in sciatic pain.

The cerebrospinal fluid almost invariably shows a pleocytosis comprised largely of lymphocytes. The protein often is increased.

The diagnosis cannot usually be made from the clinical symptoms alone. The diagnosis is often aided by a good history of exposure to the disease and can be substantiated by proper agglutination tests.

Spirochetes

A number of spirochetal infections involve the nervous system, such as syphilis, relapsing fever, rat-bite fever, and leptospirosis. Of these, only syphilis of the nervous system occurs with sufficient frequency to warrant discussion. There are three main types of central nervous system syphilis: general paresis, tabes dorsalis, and meningovascular syphilis. The clinical manifestations are generally well known and will only be discussed briefly at this time.

General paresis. The most frequent age of onset is from 35 to 50 years. The neurologic involvement is limited chiefly to the nerve cells of the frontal and temporal cortex.

The diagnosis should always be suspected in an adult showing one or a combination of the following symptoms:

1. Insidious development of intellectual and memory defects associated with impaired judgment

2. Changes in personality (irritability, reversal of character, lying, stealing, or carelessness)

3. Acute mental changes (agitation, depression, and euphoria) associated with unreal bizarre content

4. Tremor of the lips, tongue, face, and hands associated with slurred speech.

The diagnosis is strengthened by the additional presence of any of the following: history of syphilis, treatment for syphilis, pupillary changes, and positive blood or spinal fluid serology.

Once the diagnosis has been established, treatment consists of 15 to 20 million units of penicillin, occasionally given in conjunction with a course of malariotherapy.

Tabes dorsalis. The basic lesions in tabes dorsalis are in the posterior columns and in the anterior rootlets. The diagnosis should be suspected in any middle-aged person presenting any of the following symptoms:

1. Visual disturbance with bilateral optic atrophy

2. Sphincter disturbances with frequency and some dribbling

3. Severe sharp paroxysms of pain involving chiefly the muscles of the legs (lightning pain)

4. Tight constricting pain around chest or abdomen (girdle sensation)

5. Impaired gait chiefly at night

6. Painless enlargement of joints of lower extremities (Charcot joint).

The diagnosis can be confirmed by the presence of findings of posterior column involvement, such as decreased or absent deep reflexes in the lower limbs, reduced testicular pain, and impaired position sense. Pupillary abnormalities as well as a positive serology are also helpful in establishing the diagnosis.

Treatment consists of 20 million units of penicillin given with a course of malarial therapy.

Meningovascular syphilis. The diagnosis of this form of syphilis is very difficult. It may present with transient episodes of dizziness, headache, and fatigue, followed by focal symptoms, such as hemianopsia or hemiparesis, or with transient cranial nerve involvement. To substantiate the diagnosis, it is necessary to have a history of syphilis or pupillary abnormalities or a positive serology.

This form of syphilis responds promptly to penicillin therapy.

Protozoa

There are a number of protozoan diseases that can infect the nervous system, such as amebiasis, trypanosomiasis, malaria, and toxoplasmosis. All of these protozoan infections of the nervous system are uncommon and therefore do not warrant discussion.

However, brief mention might be made of toxoplasmosis. This infection occurs chiefly in infants who apparently become infected from the mother. The source of the human infection is not known, but *Toxoplasma* has its reservoir in rodents and birds.

In infants, the most diagnostic features of the disease are chorioretinitis and intracranial calcifications, which are often bilateral and may be present in the brain at the time of birth. As the child develops, he may show definite retardation, convulsions, and even focal symptoms indicative of brain damage. The cerebrospinal fluid usually contains a high protein content and a moderate mononuclear pleocytosis. *Toxoplasma* has been found on spinal fluid smears.

Helminths

Although a large number of helminths infect man, they rarely invade the nervous system. For this reason, only a few of the infections caused by these parasites that have been reported in recent literature will be mentioned.

Schistosomiasis. This infection is caused by a species of flukes that spends part of its life cycle in snails. The human infection is acquired by contact with the free-swimming forms in water in certain contaminated areas of the Pacific. Many cases of infection have occurred in military personnel who have been in the Pacific, specifically in the Leyte area.

Invasion of the nervous system results in a tumor-like granuloma which produces the symptoms of a space-occupying mass. The cerebral manifestations consist of seizures, hemianopsias, hemiplegias, and so on. The diagnosis should be suspected in any patient developing progressive focal symptoms who has been in the Leyte area.

Treatment consists of surgical removal of the granulomatous mass.

Cysticercosis. This pork tapeworm infestation is usually obtained by eating uncooked or poorly cooked meat. Once the larvae invade the body, they chiefly involve the muscles but seem also to have a predilection for the nervous system.

The clinical manifestations of nervous system involvement is kaleidoscopic, varying with the manner and intensity of the cysticerci invasion. Generally, the physician is forced to seek carefully for additional aids in the diagnosis, such as: (1) occurrence of cysticerci in the skin, muscles, or eye; (2) calcified parasites in the brain visible on roentgenograms; (3) cysticerci in the spinal fluid; (4) eosinophilia; and (5) serologic tests.

Yeasts and molds

Coccidioidomycosis. This is an endemic disease occurring in southern California, Arizona, New Mexico, Argentina, and Hawaii. The organism enters the body through the lungs or skin and produces a chronic meningitis in man.

The primary infection results in malaise, anorexia, headache, fever, cough, and joint pain. When the meninges become involved, headache, cervical rigidity, lethargy, and even papilledema develop. The disease usually extends over a period of months, producing the syndrome of a chronic lymphocytic meningitis resembling tuberculous meningitis.

The diagnosis should be suspected if the patient has resided in areas endemic for this disease. The organism often can be isolated from the spinal fluid on Sabouraud's medium. Confirmatory diagnosis may be made by injection of infected material into a guinea pig and by skin tests.

Torulosis (cryptococcosis). This is a worldwide infection, with the portal of entry in the respiratory tract. The source of the infection is probably contaminated soil. The infection may involve the lungs, skin, lymph nodes, liver, and spleen but seems to have a predilection for the brain and the meninges.

The symptomatology varies with the nature and extent of the pathologic changes. The predominant symptoms are referable to the nervous system and consist of intermittent headache, vertigo, and stiff neck. As the illness progresses, irritability, mental confusion, cranial nerve involvement, and signs of increased intracranial pressure develop. The disease advances to a state of emaciation and severe disability, but remissions may occur and last for months.

The spinal fluid is often under increased pressure and contains several hundred mononuclear cells. The protein is elevated and the sugar is reduced. Yeastlike cells may be visible on direct examination of the fluid. The organisms are readily identified on India ink preparations.

The diagnosis can be aided by intraperitoneal inoculation of white mice with spinal fluid.



Neil S. Dungay, M.D.

W. E. WILSON, M.D.

Northfield, Minnesota

WHEN DR. NEIL S. DUNGAY passed away on September 19, 1958, Northfield, Minnesota, lost one of its most respected citizens and Carleton College one of its oldest and most beloved teachers. His death was a personal loss to a host of friends and former students throughout the country. He had taught at Carleton from 1907 until his retirement in 1952, longer than any other faculty member.

While he was still actively engaged in teaching, in 1952, he first became ill with heart disease. In 1954, he suffered a stroke that caused partial paralysis of one side. During the next four years, while he was lovingly nursed by his wife, he maintained a keen interest in medical and college affairs; a second stroke in September 1958 resulted in his death two weeks later.

Dr. Dungay was born in Cannon City, Rice County, Minnesota, on September 28, 1882, the son of Moses L. and Ida M. Dungay. Following his secondary schooling, he attended the University of Minnesota, from which he graduated in 1904. He was a science teacher in the high school at Marshall, Minnesota, for several years and went to Carleton in 1907 as professor and head of the biology department. In 1913, after a year of study at the University of Chicago, he received his Ph.D. degree. While there, his eyes troubled him considerably, and for many months it was necessary for his wife to read his lessons to him.

From Chicago he returned to his teaching at Carleton, which was successful and outstanding for many more years.

In the early 1920's, Dr. Dungay attended medical school at the University of Minnesota, from which he graduated in 1925. He served his internship at the University Hospital and was a physician in the Student Health Service. After he completed his medical work at the University, he returned to Carleton, where he organized and headed the student health service. After some years as college physician, he became chairman of the newly organized Department of Hygiene and Public Health and also professor in the zoology department.

While at the University, he became a member of Phi Beta Pi medical fraternity and was elected to Alpha Omega Alpha, the medical honor society. He was a member of Phi Beta Kappa, national scholastic society; of Sigma Xi, science honor society; of the Rice County and Minnesota State Medical societies; and of the American Medical Association. He was a Fellow of the American Association for the Advancement of Science, a Fellow of the American Public Health Association, and president of the North Central section of the American Student Health Association. His biography appears in *Who's Who in America* and in *American Men of Science*.

While teaching at Carleton, he spent several summers at Woods Hole biological laboratories in Massachusetts to increase his knowledge of biology and to associate with some of the country's finest scientists. He became a life member of the Woods Hole corporation.

Dr. Dungay's first marriage was to Maude Belle Dodge, who died in 1927. In 1928 he married Freda Eyrich of Le Sueur, who survives him.

Dr. Dungay had many interests besides his professional work, to which he contributed much time and energy. He was a member of the First Congregational Church in Northfield and served several terms on the board of trustees. He assisted the former pastor of that church, the late

Edwin B. Dean, in organizing the Boys Brigade and the Boy Scout work of fifty years ago.

He was a devoted Mason, being a member of several of its bodies, a Past Master of Social Lodge in Northfield, and Grand Master of the Minnesota Grand Council of Royal and Select Masters. In his earlier years in Northfield, he was a member of the National Guard, serving as private, sergeant, and lieutenant. He was an instructor in the Student Army Training Corps at Fort Sheridan in 1918, during World War I, and was active in the Medical Procurement and Assignment Service in World War II.

Dr. and Mrs. Dungay traveled considerably, including a trip to Africa and Asia. He enjoyed reviewing these trips by showing to his friends the many pictures he had taken. Enlargements of many of them, showing faraway places, adorn the walls of his study.

Neil Dungay grew up on a farm near Cannon City and attended high school in Faribault. Mr. Alfred H. Bill, of Princeton, New Jersey, recalls those early days and his associations with Neil.

He seemed to regard the world with a kind of humorous tolerance which might become sardonic when things went wrong but was generally kindly. This quality, I imagine, never failed him. Certainly I recognized it when I last saw him, when those strokes had disappointed all his hopes of a happy and busy retirement.

As was necessary in those days, youngsters learned to do many things. Neil learned the carpenter trade, and, when it came to finishing off a house or doing a neat job of cabinet work, there was none who could do it better. He learned to lay brick, and no one did it more accurately, as is demonstrated in the outdoor fireplace of his home which he built in the later years of his life. He learned to survey, to farm, and, when it came to turning out good food on a cook stove or over a camp fire, nothing ever tasted as good.

He knew the trees, the flowers, the birds, and the tracks of wild animals in the snow. It was a real privilege and pleasure to spend days and weeks in the woods or on canoe trips with this man, so versatile in every way, so tolerant of those who were attempting to absorb some of the knowledge that he imparted. Perhaps this is one of the reasons he was so popular with young people. He was very modest, yet his associates were constantly surprised at something new that he would do or say which revealed the depths of his knowledge. One of his colleagues at the University once remarked, "I never saw anyone who knew so much about everything as Dr. Dungay."

Because of his general competence in almost any field, he was frequently called upon to do extra things which no one else seemed to be able to do. One spring at Carleton, it was thought that it would be a good idea to have a course in bird study. And who else but Dr. Dungay should be selected to teach it? He got the students up at five o'clock in the morning every day, then roamed the fields and woods for two hours identifying birds.

When the United States entered World War I in the spring of 1917, an "army" was formed of nearly all the boys in college; it was Dr. Dungay who was called to instruct the young men in the rudiments of drill and marching, because of his previous experience in the National Guard.

President Lawrence M. Gould of Carleton pays a fine tribute to Dr. Dungay:

Dr. Neil Dungay served Carleton College over a longer period of time than anyone who has ever been associated with it. He served the college in a variety of ways—as teacher, as physician, and always as a scholar and friend.

Carleton has had few men on its faculty who were as broadly trained to carry out their varied tasks as was Dr. Dungay. He was both a Ph.D. and an M.D., which eminently fitted him for his work here.

In his earlier days, Dr. Dungay was called upon to teach a variety of subjects extending beyond those of his specialties. The first year at Carleton he taught a course in geology, and from that single class there came 4 men who went on to graduate work and careers in geology. This is an indication of the stimulating character of his teaching which remained with him throughout his life.

To us who had the privilege of knowing him as a colleague, he was always generous, helpful, and most thoughtful.

To those who were privileged to take his course in general biology, it was an experience never to be forgotten. Men and women who attended Carleton over forty years ago still recall his lectures, so well organized and so clearly delivered, leading his listeners from the study of the amoeba, in the opening days of school in September, up through the evolutionary scale to the vertebrates, in May. These former students repeat over and over that it was one of the best college courses they ever attended.

Professor Leal A. Headley, who was a fellow teacher on the faculty at Carleton, says of Dr. Dungay:

To qualify himself as a college teacher, he earned a Ph.D. in biology at the University of Chicago. To add breadth and vividness to his teaching, especially in the area of physiology and hygiene, he completed a full course at the University of Minnesota Medical School.

This preparation, supplemented by lifelong contacts with the literature and the explorers in the vast field of his professional interests and combined with his reverence for life, his insistence on precision in both laboratory technique and logical inference, his clarity in presenting intricate details, and his capacity for personalizing the

individuals in his classes, made Dr. Dungay an extraordinarily good teacher.

When one has great respect and holds a person in high honor, it is difficult to find words to truly express this feeling. In my humble opinion, Dr. Dungay was the greatest teacher under whom I had the opportunity to study. It was a rare privilege. He made the most difficult easy and could present a scientific problem in such a form that all could understand it. I believe that to be a great teacher one has to be a great person, and that requirement Dr. Dungay filled to overflowing. He gave unselfishly of his time. He did not know the meaning of office hours. He was kind, calm, unselfish, understanding, and very patient.

MRS. IRENE QUINN HAWKINSON, Carleton class of 1925
Former student and associate teacher of Dr. Dungay

When I was at Carleton, he was regarded as a superb teacher, and his leaving to take his M.D. degree was a definite loss to the College temporarily. When he was at the University Medical School, he was the class yardstick of academic excellence.

He took an active part in the development of the public health activities of Carleton College and, when called upon to do so, presented excellent medical papers, maintaining an erudite knowledge of medical progress though quite removed from personal practice.

DEXTER LUFKIN, M.D.
Hot Springs, South Dakota

The best evidence of the impact of his personality, inspiration, and ability to communicate is the extraordinary extent to which one can picture him in the mind's eye standing before the class in Laird Hall. I can see his face and even the way he dressed as he illustrated his points on the blackboard and shared with us his wide scholarship. Dr. Dungay had the remarkable gift of mak-

ing each student feel that he was talking directly to him as he lectured to the entire class. I can visualize him, too, during laboratory sessions, seminars, and informal groups. Always there was the feeling of warm, friendly, personal interest.

The empiricism of Dr. Francis Peabody of Harvard that 'the first principle in the care of the patient is the care for the patient,' could be paraphrased, 'the first principle in the care of the student is the care for the student,' as applied to Dr. Dungay. He really cared about what happened to us then and in our subsequent careers.

When alumni returned to Northfield, they could always count on a first name greeting from Dr. Dungay and a deep interest in their accomplishments. They could always rely on replies to letters and cards from his or Mrs. Dungay, even during the latter painful years of growing ill health.

Those who had the good fortune to come under Dr. Dungay's warm-hearted and inspiring influence will continue to pass along the torch he lighted for us.

JEAN CURRAN, M.D.
Boston, Massachusetts

Dr. Dungay was one who, in spite of his great scientific achievements, was a very humble and friendly man. He was modest in his accomplishments, making friends in every field. He will be sincerely missed by many, not only as a successful man but as a friend whom one regrets to lose.

NORMANLENDE, M.D.
Faribault, Minnesota

Although Dr. Dungay has died, he continues to live in the lives of hundreds of former students, who strive to emulate his example as teacher, physician, and good citizen.

Transactions of the North Dakota State Medical Association

SEVENTY-THIRD ANNUAL MEETING

Grand Forks, North Dakota, April 30, May 1, 2, and 3, 1960

(Continued from the November Issue)

Report of the Reference Committee on Resolutions

Dr. F. A. DeCesare, chairman of the committee, presented the following resolutions.

RESOLUTION

Whereas, the members of the North Dakota State Medical Association attending the seventy-third annual meeting of the Association in Grand Forks have enjoyed the hospitality and kindness of the fair city, and

Whereas, the mayor of Grand Forks and his associates, the press, radio, hotels, and businessmen have made this session a memorable one,

Now, therefore, be it resolved that the House of Delegates express its appreciation by directing a copy of this resolution to the Honorable Mayor of Grand Forks.

This resolution was adopted.

RESOLUTION

Whereas, the exhibitors have shown great effort and interest in this meeting and former meetings in developing their exhibits and adding to the scientific interest,

Now, therefore, be it resolved that the North Dakota State Medical Association extend to them our hearty welcome and thanks, and

Be it further resolved that a copy of this resolution be sent to each exhibitor.

This resolution was adopted.

RESOLUTION

Whereas, Dr. John C. Fawcett, president of the North Dakota State Medical Association for the year 1959-1960, has given untiringly and unselfishly of his time and services toward the continued progress of medical practice in North Dakota,

Now, therefore be it resolved that the assembled delegates show their appreciation by a rising vote of thanks.

A rising vote of thanks adopted this resolution.

Continued on following pages

RESOLUTION

Whereas, the members of the seventy-third annual meeting of the North Dakota State Medical Association have thoroughly enjoyed and profited by an excellent scientific program, and

Whereas, the host, the Third District Medical Society, and the various chairmen and committeemen have excelled in providing the members of the association with the niceties of a gracious convention,

Now, therefore be it resolved that the assembled delegates demonstrate their appreciation by a rising vote of thanks.

A rising vote of thanks adopted this resolution.

RESOLUTION

Whereas, the Woman's Auxiliary to the North Dakota State Medical Association has, through various projects entailing continuous work and effort, raised the sum of approximately \$18,000 for their medical Student Loan Fund at the Medical School of the University of North Dakota and donated \$432.71 to the American Medical Education Fund, and

Whereas, these funds have been of great value to many medical students and to the medical school, and

Whereas, participation in these and other worthwhile projects has won them meritorious certificates from the American Medical Educational Foundation and *Today's Health* and has all made for continuing good will for the medical profession in North Dakota,

Now, therefore be it resolved that the House of Delegates of the North Dakota State Medical Association convey to the Woman's Auxiliary of the association its appreciation and thanks for their excellent work, and

Be it further resolved that a copy of this resolution be directed to the president of the Woman's Auxiliary.

This resolution was adopted.

RESOLUTION

Whereas, Dr. Leonard Larson, a trustee of the American Medical Association; Dr. Willard Wright, an delegate to the American Medical Association; and Dr. C. J. Gaspel, secretary of the North Dakota State Board of Medical Examiners, have done memorable service and brought honor to the North Dakota State Medical Association,

Now, therefore be it resolved that this association take cognizance of their services and pay tribute to these men for their efforts on behalf of the North Dakota State Medical Association, and

Be it further resolved that a copy of this resolution be forwarded to each of these men.

A rising vote of thanks adopted this resolution.

RESOLUTION

Whereas, many physicians believe and various studies have shown that the goals, purposes and accomplishments of the medical profession with regard to scientific, socioeconomic, and political developments in the field of medical care are poorly understood by the public, and

Whereas, greater public understanding and support are necessary if the best medical practice is to survive and continue to serve the public, and

Whereas, an effective public relations program is the best insurance that the purposes and performance of the medical profession will be understood and appreciated by the people of our state,

Now, therefore be it resolved that the North Dakota State Medical Association activate its program to expand and accelerate public relations through the North Dakota State Medical Association's Public Relations Committee.

The Committee on Resolutions recommended a change in this resolution, namely, that the last three words be changed from Public Relations Committee to elected officers of the association.

This resolution in its revised form was adopted.

RESOLUTION

Whereas, it is the time-honored ethical practice of the physician to aid all who are in need of medical care; and

Whereas, the physician does not turn away the person in straightened financial circumstances;

Now, therefore be it resolved that the members of the North Dakota State Medical Association reiterate once again through the association's ruling body, the House of Delegates, that no person in North Dakota is ever refused medical care based on inability to pay.

This resolution was adopted.

RESOLUTION

Whereas, during the past half century, scientific advances and improvement in community health services, as well as in the standard of living, have helped push back tuberculosis in the United States, and

Whereas, the United States Public Health Service and National Tuberculosis Association asked a small group of national leaders

in several phases of public health and tuberculosis control to define the major gaps in present practices and to suggest the action needed, and

Whereas, the major recommendation of this Arden House Conference on Tuberculosis, held at Harriman, New York, November 29 to December 2, 1959, was "The Ultimate Goal is Elimination of Tuberculosis in the United States of America"; and

Whereas, the North Dakota Tuberculosis and Health Association feels that this goal can be reached by mobilizing all resources for a widespread application of the scientifically demonstrated and medically accepted procedures of adequate chemotherapy,

Now, therefore be it resolved by the North Dakota State Medical Association that the recommendations of the Arden House Conference on Tuberculosis deserve the cooperation of every physician in North Dakota, and

Be it further resolved that public health authorities assume their responsibility for seeing that all tuberculous patients receive adequate treatment and that competent medical, nursing, and rehabilitation care of the tuberculous patient be the goal of every North Dakota physician and that the North Dakota State Medical Association actively support this program of tuberculosis elimination.

This resolution was adopted.

RESOLUTION

Whereas, almost 500,000 persons are accidentally poisoned in the United States annually, and

Whereas, daily almost 1,000 children under the age of 5 years are accidentally poisoned by consuming household products containing harmful chemicals, and

Whereas, household products sometimes are not adequately marked so as to warn of their poisonous properties, with the result that they sometimes are not stored out of the reach of children, and

Whereas, there is a great need for the education of the American people as to the dangers of accidental poisonings and the need to keep from the reach of children all household products which contain harmful chemicals,

Now, therefore be it resolved by the Sixth District Medical Society that the North Dakota State Medical Association be requested to encourage the people of North Dakota to learn of the dangers of accidental poisoning and to take preventive measures, and

Be it further resolved that the present labeling law of North Dakota be amended to include all types of household poisons.

Speaker Dodds asked for a discussion on this resolution, as it was not clear to the Chair how it would be carried out.

Dr. DeCesare stated that the committee thought that a proper committee could work with the state and recommend to it that this Association feels that some changes should be made in the labeling law.

Dr. Tudor advised the delegates that we now have a model labeling law in the hands of a bipartisan committee, and it will help to bring to the attention of the state the fact that the association feels that changes should be made.

This resolution was adopted.

RESOLUTION

Whereas, the North Dakota State Medical Association has never opposed the care of service-connected illnesses in Veteran Administration Hospitals, and

Whereas, North Dakota State Medical Association has not opposed the treatment of veterans in Veteran Administration hospitals if these people were indigent, and

Whereas, in recent years more and more nonservice-connected illnesses have been treated in Veteran's Administration hospitals, and

Whereas, the great majority of these patients are responsible citizens with steady employment and should assume the same responsibilities for their medical care as do nonveterans,

Now, therefore be it resolved that the North Dakota State Medical Association join with over 25 other state societies in opposing the continued admission of patients with nonservice-connected illnesses to Veterans Administration hospitals, and

Be it further resolved that there be congressional investigation of this unnecessary spending of tax money, and

Be it further resolved that a copy of this resolution be sent to the American Medical Association and to the executive secretary of the State Medical Association of Maryland.

This resolution was adopted.

RESOLUTION

Whereas, the present day and projected needs of medical education are well known to all of us, and

Whereas, the members of the North Dakota State Medical Association have shown their willingness to support medical education through the American Medical Education Foundation, and

Whereas, it has been proposed that physicians in the state could enlist the administrative services of Blue Shield in making their contribution to medical education, and

Whereas, the proposed approach would provide an avenue of contributing that might prove more convenient to some physicians and thereby increase the number of contributors as well as the amount given to medical education,

Now, therefore, be it resolved that the North Dakota State Medical Association approve the use of Blue Shield administration to assist and encourage the physician to make a voluntary contribution to the American Medical Education Foundation.

This resolution was adopted.

Dr. De Cesare advised the House that the last resolution would be taken up under the heading of other business. This resolution, therefore, was postponed.

Dr. De Cesare thanked the members of his committee for their willingness to work. He moved that the report be adopted as a whole. Dr. Mahowald seconded the motion, and the report as a whole was adopted.

F. A. DE CESARE, M.D., Chairman

R. E. MAHOWALD, M.D., Vice-Chairman

MILTON NUGENT, M.D.

J. N. ELSWORTH, M.D.

M. W. GARRISON, M.D.

Speaker Dodds requested 5 members of the House to volunteer to escort the 50-Year Club members to the rostrum on Tuesday afternoon and asked that they give their names to Mr. Limond.

The next order of business was the fixing of the per capita dues for the ensuing year. Speaker Dodds called on the chairman of the Resolutions Committee to return and present the resolution which was tabled the preceding day. It reads as follows:

RESOLUTION

Whereas, the importance of public education to combat the passage of additional social legislation is well recognized by each physician, and

Whereas, the Council has recommended that a public relations program be developed by the North Dakota State Medical Association,

Now, therefore, be it resolved that the annual dues of the North Dakota State Medical Association shall be increased in the amount of \$25 per member, and

Be it further resolved that the money so obtained shall be used exclusively for the purposes of public information, public relations, and legislation.

Dr. DeCesare moved that the resolution be adopted. Motion was seconded by Dr. Baumgartner.

The question was raised of who will control the expenditures of the additional fund. The Chair called on Dr. John Fawcett to amplify the answer to this question.

DR. FAWCETT: "The form of that particular resolution was not entirely clear to me as to who would use the money. The Council, of course, has control of the expenditures of the association. Let me digress from the question for just a moment.

"I want to express my very great appreciation to each and every one of you who helped to make this year what it was. We found that the greatest difficulties were in the field of public relations and legislative activities. I do not think these two things can be separated. Public relations and legislative activities on a national level have been very active but inadequate. It has been brought home to us that we have to carry on at the grass roots level. It becomes essential, of course, that the relations of each individual to the public and to the press, TV, and radio be of the very best. It is essential that we know our legislators personally in order to change their thinking. This is true with the year ahead. We know that we need much more work in public relations and that such work has to be directed. The Council discussed this at

great length with regard to the feasibility of hiring a full-time public relations man to implement this program. This would also necessitate having a responsible committee on public relations which would be directly responsible to the Council.

"The Council would have control of the committee, of course, and it would also be coordinated with the activities of the Blue Cross-Blue Shield. The second requirement is just plain money. The only way we can implement this program is by substantially increasing the dues. Right now your association is just about breaking even from its income and its expenditures."

Dr. Peters next addressed the members of the House as follows: "I feel that this is an opportunity for the House of Delegates to have a free and open discussion here today. I think that when you go back to your districts, you should be able to tell the members how you came to certain conclusions at this meeting. It is obvious that our state office in Bismarck, with only Mr. Limond and a stenographer, cannot handle an additional load. The staff must be increased. The legislature is meeting in January. We have the public relations problem that came out in the newspapers in Jamestown. We need a proper public climate. I feel that this problem of dues is stressed too much. I do not think that we, as physicians, are going to be hurt by a \$25 increase in dues per year. It is tax deductible. For one, I would like to see this resolution passed and the increase in dues granted, so that this program of public relations can be brought into effect. It is going to need guidance. It should get off to a slow start. We should find our way slowly. It should have some active direction, and I think that the House has confidence that its officers will see to it that it is properly directed."

Dr. Wright advised the House that the Association will have available, through the American Medical Association, a man completely experienced in this sort of thing, who would be able to devote a week or more of his time to come to North Dakota and give us the information we need to set up a program of this kind.

Dr. DeCesare addressed the House next, as follows: "I would just like to bring out a couple of points. What are we going to tell our colleagues when we get back home? What is going to be done with this increase in dues? There are those who are opposed to a high salaried public relations man. We feel that this money should be endorsed to the Council and officers and let them use their discretion. However, I have heard from several people who feel that adopting a resolution of this type, which asks for an increase in dues for public relations alone, may not set well with the lay people and the press at this time. I do not know. We thought that this increase could be handled for the purposes of public information, and then people could not say that it is entirely earmarked for one thing only."

The motion passed, the resolution was adopted, and the increase in dues was granted.

The Chair echoed Dr. DeCesare's remarks by stating that every delegate should present this resolution to their members at the next district society meeting, so that the members understand it thoroughly.

The next order of business was to choose a city in which to meet in 1963. In 1961, the meeting will be held in Fargo. The House of Delegates previously approved having the seventy-fifth anniversary meeting in Bismarck, the dates to be worked out with the South Dakota Association. The Chair asked the House for an opinion in regard to holding the meeting in Bismarck on June 2 to 6, 1962, which is a change from the meeting

date of the first week end in May. Contact has been made with the South Dakota group, and the Chair asked that someone make a motion to the effect that the meeting dates be set for June 2 to 6 for the seventy-fifth anniversary meeting in 1962. Dr. Tudor so moved, and Dr. Macaulay seconded the motion. The motion passed.

Dr. Naegeli, representing the Fourth District Medical Society, tendered the North Dakota State Medical Association an invitation to meet in Minot in 1963. Dr. Palmer moved that this invitation be accepted, and Dr. DeCesare seconded the motion. The motion carried.

Report of the Nominating Committee

Dr. F. A. Hill, chairman, gave the following report:

President	Dr. C. M. Lund, Williston
President-elect	Dr. E. H. Boerth, Bismarck
First vice-president	Dr. E. J. Larson, Jamestown
Second vice-president	Dr. A. R. Gilsdorf, Dickinson
Speaker of the House	Dr. G. A. Dodds, Fargo
Vice-speaker of the House	Dr. C. H. Peters, Bismarck
Secretary	Dr. Wm. Buckingham, Elgin
Treasurer	Dr. R. D. Nierling, Jamestown
Delegate to the AMA	Dr. W. A. Wright, Williston
Alternate delegate to the AMA	Dr. T. E. Pederson, Jamestown

Councillors (term expiring in 1962—to complete the unexpired term of Dr. R. O. Goehl):

Third District	Dr. P. H. Wontat, Grand Forks
Fourth District	Dr. F. D. Naegeli, Minot
Fifth District	Dr. G. Christianson, Valley City
Tenth District	Dr. K. G. Vandergon, Portland

Board of Medical Examiners (term expiring in 1961—to complete the unexpired term of Dr. D. J. Halliday):

Dr. V. J. Fischer, Minot

Terms expiring in 1963:

Dr. C. J. Glaspel, Grafton

Dr. R. C. Painter, Grand Forks

Dr. J. D. Craven, Williston

Dr. Mahowald moved that the nominations cease and the secretary be instructed to cast a unanimous ballot for the foregoing slate of officers. Dr. Hammangren seconded the motion, and the motion passed.

Dr. Lommen next addressed the House, stating that the delegates from the Sixth District had been advised and instructed to see what they could do about a problem that doctors are having with the Workmen's Compensation Bureau. A letter had been received from a member of the sixth district, in which he cited 3 cases which he thought were not treated properly, and he stated that he did not get the consideration from the Workmen's Compensation Bureau that he thought he should have. Dr. Lommen recommended that the Council take the matter up at its next meeting.

Dr. Dodds instructed Dr. Lommen to communicate this material to the chairman of the Council.

The next item of new business was the resignation of Dr. Gilsdorf as councillor of the Ninth District because he had been elected to the office of second vice-president.

Dr. Hill, chairman of the Nominating Committee, offered the following nomination for the office of councillor to fill the unexpired term of Dr. Gilsdorf: "We wish to present the name of Dr. Keith Foster of Dickinson for nomination for councillor to fill this unexpired term of Dr. Gilsdorf." Motion was seconded by Dr. Hankins and carried. Dr. Keith Foster was elected to the office of councillor from the Ninth District.

Dr. Van der Linde next proposed that officers from the second vice-president on down should not be re-

quired to resign from other offices that they might be holding at the same time. Motion was seconded by Dr. Tudor and carried.

Dr. Baumgartner asked the House for a clarification of the following: "The president stated in his report that during the last annual meeting in Bismarck, we were confronted with complete disruption of the medical organization at the State Hospital in Jamestown. We offered to be of assistance in obtaining and securing new psychiatrists and staff, which offer was accepted (apparently with gratitude) by the governor and the chairman of the State Board of Administration. An Advisory Committee was appointed, with Dr. Joseph Sorkness as chairman, who reports to me that Mr. Joos, chairman of the Board, has never seen fit to use or consult the committee. I understand that replacements of the new staff are pretty well completed but that (1) qualifications of the new staff were never screened by our committee and (2) a nonmedical superintendent still heads the institution."

Dr. Baumgartner then stated that he noted the Committee on Mental Health reported as follows: "They did not meet formally this year, since the state president appointed a 3-man Liaison Committee to work with the State Board of Administration. It was felt best to allow these men to act without interference. They have managed to aid in getting some psychiatric personnel at our State Hospital."

According to the last report, Dr. Baumgartner believed that the 3-man Liaison Committee had been active, and he asked whether there was a conflict or if he misunderstood.

Speaker Dodds answered this as follows: "Perhaps I, as a member of the committee, can speak a little on this. The Liaison Committee was appointed with Dr. Sorkness as chairman and Dr. Boerth and myself as members. During the past year, we met on 3 occasions with the Board at Jamestown. It is my feeling that certain steps of progress have been made. It is also my feeling that the Board does appreciate the problem. There is still a division of authority. There is a medical director and a business administrator. We recently tried to obtain a very qualified doctor of medicine to head this institution, and we had authority to do so. Dr. Sorkness contacted this man, but by the time we could interview him, he had been employed elsewhere. There is a complete medical staff of foreign extraction. That is all I can add. Maybe Dr. Boerth can add something further."

Dr. Boerth stated that the governor was informed of the last meeting and given the minutes of it.

Speaker Dodds recommended that all of the delegates read the *Handbook*, since a great deal of work had gone into it.

The motion was made, seconded, and passed for adjournment. Meeting adjourned at 4:30 P.M.

Scientific Program

May 2, 1960

Grand Forks Armory-Auditorium

8:30 to 9:15 A.M. Registration

9:15 to 9:30 A.M. Greetings from the mayor of Grand Forks and the president of the Grand Forks District Medical Society.

9:30 to 10:00 A.M. "The Use of the Laboratory in Modern Medicine," W. E. Cornatzer, Ph.D., M.D., Grand Forks.

10:00 to 10:45 A.M. "Cardiac Aspects of Chest Pain," Alan Rusterholz, M.D., St. Paul.

10:45 to 11:30 A.M. Intermission

11:30 to 12:00 noon "The Neurological Psychosomatic Aspects of Chest Pain," Gordon B. Kannan, M.D., St. Paul.

NOON RECESS

1:30 to 2:45 P.M. "Endometriosis," Leonard Lang, M.D., Minneapolis.

2:15 to 3:00 P.M. "Indications for Heart Surgery and the Treatment of Cardiac Arrest," Morley Cohen, M.D., Winnipeg (sponsored by the North Dakota Heart Association).

3:00 to 3:30 P.M. Intermission.

3:30 to 5:00 P.M. Tour of facilities at the Rehabilitation Center with demonstration of physical therapy technics.

May 3, 1960

Grand Forks Armory-Auditorium

8:30 to 9:00 A.M. Registration.

9:00 to 9:45 A.M. "Aortography," James B. Murray, M.D., Fargo.

9:45 to 10:30 A.M. "Diabetic Emergencies," Howard Root, M.D., Boston.

10:30 to 11:15 A.M.—Intermission.

11:15 to 12:00 Noon "Current Surgical Procedures for Carcinoma of the Rectum," Edward S. Judd, M.D., Mayo Clinic, Rochester (sponsored by the North Dakota Division of the American Cancer Society).

NOON RECESS

1:30 to 2:30 P.M. Presidential address, J. C. Fawcett, M.D., president, North Dakota State Medical Association.

Introduction: honorary members of the North Dakota State Medical Association.

Inaugural address: C. M. Lund, M.D., president-elect, North Dakota State Medical Association.

2:30 to 3:15 P.M.—"Current Plastic Surgical Concepts of General Interest," John K. Grotting, M.D., and Lyle V. Kragh, M.D., Minneapolis.

3:15 to 4:00 P.M. Intermission.

4:00 to 4:45 P.M. "The Newer Tranquilizing and Related Drugs," Wilbur Benson, M.D., Minneapolis

PRESIDENTIAL ADDRESS

Dr. J. C. Fawcett

It is with mixed emotions that I come to the end of my term as your president. I can only hope that in some small way I may have contributed my bit.

As I review the events and problems of the past twelve months, as well as those of the past few years, it would seem that they might be likened to growing boys. For a few months, the boy will seem to shoot up in height; then, for a few months, no change; then again, a rapid growth in stature; and so on in spurts until he has achieved manhood. But with his physical growth come his problems and trials of that particular age and time, becoming more involved with the years and seemingly never completely resolved. Would you not say that such might be the case with medical groups—local, state and national?

Originally, the State Medical Association was adequately staffed by officers from amongst its members. As the administrative load increased, it was found necessary, just after World War II, to secure the services of a part-time executive secretary. The load further increased, and it became advisable to have a full-time executive secretary, who, in turn, must have a secretary-stenographer to do the job right. Their work entails much correspondence, liaison for better understanding and better relations with other groups, close watch over all impending legislation, public relations work, and so forth.

In the past twelve months, the volume of work entailed in keeping current and keeping medicine solvent, so to speak, in the various areas has grown by leaps and bounds. This naturally increases the administrative work load proportionately in both the Bismarck offices and in the many committees. These various committees are now, in many instances, donating more of their time than they can well afford away from their own private practice. On some committee chairmen, it must be truly a burden. I anticipate very soon the need for more full-time paid personnel in our Bismarck office.

I recently read a definition of committee, which is: "The unable who have been asked by the unwilling to

do the unnecessary!" Such, I can assure you, is not the case with your state society committees. The big majority of chairmen and members are really dedicated men who have given many days of their time, driven many hundreds of miles, paid most or all of their own expenses, and have deliberated long over the controversial problems confronting them. Many have gone to meetings out of the state, again at their own expense.

You may well wonder what I am leading up to in such a roundabout fashion. What is there about the present and future that necessitates the huge increase in work of which I have been speaking? As I envision it, there is *nothing new*—just a new slant, a new importance to the old. We have been too complacent and allowed things to happen that should never have happened. Through this complacency and diffidence, we have allowed forces to gain the ascendancy that can well set medicine back many years. We have been so satisfied with the unprecedented advances of scientific medicine that we have had a tendency to develop a superman attitude and have lost that old, close, family practitioner type understanding that used to give the physician influence and prestige in the eyes of his patients, neighbors, local groups and organizations, church, and so forth. With this loss of what is now called public relations, we have allowed groups, institutions, and organizations which would like to take over, control, and dictate to medicine a chance to come very close to this very thing.

We see some remark derogatory to medicine in the paper; it is not contradicted. A public official comes out with the avowed intention of passing legislation to take over or control medical practice; he makes public statements to that effect, which are heard or read by his constituents—and, what have we done to neutralize his statements.

I do not say that we are standing still and that no progress has been made. For a number of years, the American Medical Association has been taking a positive approach to legislative and public relations problems and has improved its, and our, situation immeasurably. Its Washington office has kept a sharp watch on all proposed legislation not only directly or indirectly pertaining to medical matters but also on bills proposed which might affect education and public welfare. Through its executive officers, it has given testimony on many of these bills at our House and Senate committee hearings. It has been responsible for helping to stave off, delay, or kill much objectionable, or even vicious, legislation.

The House of Delegates of the American Medical Association has had many decisions to make which directly affect the future welfare of medicine. All of us may not have agreed with its decisions and conclusions, some of which were compromises on controversial issues, but it studied them long and made the decisions that it thought were best for all. Both the administrative and legislative officers and committees have known, and have so far not quite succeeded in convincing the individual physician, that all bad legislation cannot be fought in Washington and on the national level. The popularity and esteem we would wish to hold cannot be established alone by the work of the AMA officers, House of Delegates, the various AMA councils, or the Chicago and Washington offices. Unless every single member of our great organization comes to the realization that a chain is only as strong as its weakest link, that the cumulative efforts of each individual physician working toward better public relations are essential, and that each one must become actively interested in legislative matters—then, and only then, will we have a truly effective force

capable of influencing public thought and legislative action. May I here further emphasize that to discuss these matter at our state and local meetings and to agree therein that all of these things are true is not sufficient! Each physician must follow through with positive action. This is action that is not a "one-shot" affair but must continue from one year to the next. To do this effectively, the physician must be well-informed, attending all local medical society meetings and reading such instructive material as may be in the journals, medical society newsletters, pamphlets, and so forth. He must be active in local community activities, such as school, church, service clubs, and the political groups of his choice.

From the standpoint of the offices of the State Medical Association, all this is perhaps striving for a medical utopia. I believe that, with an all-out effort, much of it can be attained. We must work not only in 1960 and 1961 but for many years to come and many generations to come, in order to build our North Dakota State Medical Association of the future.

Introduction: Honorary Members 50-Year Club Members

DR. J. C. FAWCETT: I now come to a very enjoyable portion of our program, which concerns itself with a small but important segment of our membership. I refer to the 50-Year Club and, specifically, to the new members of this club to whom it is my pleasure to present today with tokens of their membership. These are the men who have seen fifty years of active medical practice, good years and bad, and have watched our association develop into what it is today.

As I introduce each of these physicians and present him with his token of membership in this club, I believe you will all join me in extending to each our heartiest congratulations and our hope for many more years of fruitful practice.

Gentlemen, I now present to you these fellow practitioners of ours, members of our State Medical Association, who practiced medicine for fifty years or more.

DR. CECIL C. SMITH, Mandan: Dr. Smith graduated from the State University of Iowa in 1910. He was born July 11, 1886, in Coon Rapids, Iowa. He was licensed in North Dakota in 1910 and practiced in Stanton from 1911 to 1918. The North Dakota State Medical Association does hereby award Dr. Smith the Certificate of Distinction in recognition of his practice of medicine for fifty years. His untiring ministry to the ill have done honor to God, his community, his profession, and himself.

Dr. McLean escorted Dr. Smith to the rostrum and gave him his pin.

DR. FRANK DEASON, Grafton: Dr. Deason was born September 22, 1887. He attended Northwestern University Medical School in Chicago and graduated in 1910. He was licensed in North Dakota in July 1910 and practiced in St. Thomas from 1910 to 1920 and since then has practiced in Grafton. The North Dakota State Medical Association does hereby award him the Certificate of Distinction in recognition of his practice of medicine for fifty years. Through his proficient and untiring ministry of the science of healing, he has done honor to his God, his community, his profession, and himself.

Dr. Hill escorted Dr. Deason to the rostrum and presented him with his pin.

DR. T. H. LEWIS, Fargo: Dr. Lewis was born November 13, 1886. He graduated from Indiana University in 1910 and has practiced medicine for fifty years. He was

licensed to practice in North Dakota in January 1916. He practiced in Sutton until 1921 and since then in Fargo. He was awarded the Certificate of Distinction for his unselfish devotion to his patients and his loyalty to the medical profession.

Dr. Painter escorted Dr. Lewis to the rostrum and presented him with his pin.

DR. H. J. MEUNIER, Oakes: Dr. Meunier was born January 24, 1883. He graduated from the University of Louisville in 1910 and was licensed to practice in North Dakota in January 1912. He was awarded the Certificate of Distinction.

Dr. Mahowald escorted Dr. Meunier to the rostrum and presented him with his pin.

DR. M. G. FLATH, Stanley: Dr. Flath was born December 28, 1882, in Ontario, Canada. He graduated from Northwestern University Medical School in Chicago in 1910 and was licensed in North Dakota in July 1910. In September 1950, a drive was put on in Stanley, and the memorial was used for a new hospital in honor of Dr. Flath. He received his certificate of Distinction.

Dr. Hill escorted Dr. Flath to the platform and presented him with his pin.

DR. FAWCETT: I would also like the families of these men who are attending today to stand and receive applause. There are also 2 other members of this club here: Wm. Witherstine and Dr. French.

It is now the day and the hour for me to step down as your president and turn over the duties of office to your new president, Dr. Carroll Lund of Williston. Dr. Lund is well known to you and will serve you well, as he has proved in his various positions on State Medical Association committees and, particularly, in his work in the Cancer Society.

My very best wishes to you, Carroll, in your work and activities for the year 1960-1961.

INAUGURAL ADDRESS

Dr. C. M. Lund

Thank you, Dr. Fawcett. Our numerous meetings during the past year have been an encouragement and an inspiration to me, and I want you to know at this time how much I deeply appreciate all of the hard work that you have done for the association not only during the past year as president but in the previous years. I hope that you will continue to give your wise counsel and the benefit of your experience to all of us who follow you. I not only want to thank Dr. Fawcett, but I wish to extend on behalf of the State Medical Association our profound thanks to all of our predecessors who have stepped up to this rostrum and accepted the responsibilities of the office of president.

I have attempted to watch rather closely the manner in which this office has been conducted. All of my predecessors had one thing in common. They have all been unalterably opposed to any plan tending to foster socialized medicine and control by government of medical practice in this country. I also am opposed to such plans.

I am also further convinced that those who are attempting to alter our existing freedom and to capture medical practice are doing so because this provides the simplest mechanism for changing the democratic philosophy upon which this country was founded and has continued to prosper. If I do nothing else during the year 1960-61, I will not destroy anything that my predecessors in office worked so hard to obtain and will further continue by thought, word, and action to oppose any federal restrictions or replacements of our present medical system.

I have not inquired, but I am sure that many who

have stepped up to this podium have presented a well thought out and organized plan for improving some departments of this society during the year of their tenure. I am no exception, but I am sure that a year hence I will find that I have initiated nothing new or important. However, during the course of my visitations to the various medical societies in the coming year, I would like to emphasize a few points which I feel are for the good of the society.

First, this will be another election year, and it appears to me that the time has come when a doctor, whether he likes it or not, must enter the political field. I mentioned just yesterday to the Woman's Auxiliary that we undoubtedly would draft the fairer sex to help gird ourselves for battle during the coming campaign. I think that almost every doctor in this state is quite aware at the present time who is for us and who is against us. Strange as it seems at times, our political friends do us unestimable harm. Promising candidates should be canvassed prior to election and their ideas on the medical and ancillary organizations should be obtained. It would be most advantageous if we had a doctor or a doctor's wife to represent us during future sessions—one in the House and one in the Senate. The sacrifice of two months away from work is a great deal to ask. However, we should be able to find someone politically ambitious and rally to his cause. During the 1961 legislative session in Bismarck, we are going to have a room or small suite available for the doctors of North Dakota at the G. P. Hotel. I would like to see every doctor come to Bismarck at least once. He would be welcome to use the facilities available and at the same time have a friendly visit with representatives and senators from his own area. The usual bills that we have to combat will again appear. We will need all of our manpower to oppose any adverse bills that appear before our legislators, and I strongly urge all of you to contact your representatives and senators in advance.

On the national front at the present time, and possibly at this very moment, important medical legislation could be in the process of enactment. The impact of the outcome may not affect us at the moment, but it will be far-reaching and I daresay that possibly in ten years' time the evil work of compulsion may be hovering over us. At the present time, the AMA is fighting our battle in Washington and deserves our full support. I was more than mildly surprised when I was informed that of the total number of doctors in the United States, only 70 to 75 per cent belong to the AMA. In North Dakota, our 1959 record was 442 members out of a potential 448, which is excellent. I am sure that you have been favorably impressed during the past year or so of the political activity emanating from the AMA office. We are being alerted constantly regarding present legislation. We are receiving more literature and news. We have direct representation in North Dakota and are most fortunate in having, we hope, a president-elect, Doctor L. W. Larson, of the AMA and also a delegate, Doctor W. A. Wright, who is a member of the AMA Council on Medical Service. Both of these men deserve our full support for the many hours that they have unselfishly contributed to our Association.

Recently, in some of the local newspapers in North Dakota, editorials and letters appeared regarding a free hospital and medical plan in one of our neighboring provinces to the north. I made a personal contact with a reliable source to determine what this plan was and what it offered. I would like to mention a few of the

impressions that I received from doctors participating in this plan.

1. At the present time, there is practically no such thing as private collections.

2. For indigent care, 40 per cent of the minimum scale is received.

3. Hospital beds that were normally available are at the present time unobtainable. One doctor informed me that he was scheduled in advance for nine months for elective surgery because he could not get his patients into the hospital.

4. Five years ago, at the onset of this plan, the cost to the government was \$5 million. Five years later, the cost of the same plan and the same set-up has increased to \$41 million, or approximately 8 times as much.

5. If this system continues, there will soon be a great exodus of doctors from the province. Mind you, this is a neighboring province, and this type of hospital-medical care could not only well be implanted on receptive minds in our state but it would not be impossible to find ourselves in a similar predicament in five years.

6. The government issues every man, woman, and child \$5 a year as a pacifier. This money can be used in any manner a person wishes—savings, education, spending, and so on.

Are the doctors sitting around placidly allowing this to continue? Their attack has evolved into a new public relations organization. There are 900 doctors in this particular area. Dues have been increased \$100 per year, the amount of which is earmarked for public relations.

This brings me into our own public relations situation. I have heard often throughout the state that our association is weak in this department. With this comment, I heartily agree. There are those in our association who might disagree with me and censor the appearance of doctors on TV, on the air, in the press. It is true that the impact on the public of opposition to any adverse medical legislation carries more weight when it is presented by someone other than a doctor or anyone connected with the medical profession. However, this does not bar the doctor himself from participating in vigorous public relations organizations. I will strongly suggest that we begin now to improve our own relations by increasing our administrative staff. Our dues should be increased by a sizeable amount in order to finance this department. I recall thirty years ago when \$1 was \$1 and mighty difficult to obtain. At that time, I was a member of a musicians' union in Chicago and paying dues of \$75 a year. It is rather a strange paradox that thirty years later I find myself a member of a medical organization and still paying \$75 dues. Various union members at the present time are paying many times more than we, and the results they are obtaining certainly are apparent.

A total gross income of the doctors in this state is being protected and administered mainly by 2 people, which seems very inadequate to me. I am sure that we all protect our lives and personal property much more than we do our own organization. All of this is necessary for the price of freedom. I have a strong impression that there are those in our profession who are not very interested in the notion of freedom and that, of those who are interested, not all appreciate what must be done to keep it. If this attitude continues, the phrase "price of freedom" will be engulfed by one word, "compulsion," which is an evil word. It carries with it the aroma of medieval times when slavery was an accepted standard of living and when the minority group dictated its will

upon the masses and used every means of cruelty known to man to make sure that slaves toed the line. Certainly, compulsion is not what we today would call progressive thinking. Today, a man compelling another to submit would be branded a bully. Society would not allow such action. The culprit would be jailed and rightly so. If the government says that it is necessary to establish a compulsory program of prepaid hospital and medical care for the people of the United States, it is adopting the methods of an ancient tyrant by telling, not asking, the individual what he needs or wants. That the medical care of the people of the United States could be made a political football would be bad enough, but to take freedom of the individual away and put compulsion in its place is nothing short of dictatorship.

I have often wondered what our sons and daughters are thinking of, especially premedics, students in medical schools, and those in ancillary medical services. A multiplicity of verbal darts thrown in our direction in current newspapers, magazines, radio, television, and other mediums of communication certainly must cause some impression on them and possibly instill great doubt as to their future. In addition, I am sure that they are made aware of the ease and accessibility of the courts in instituting action against our profession. This certainly does not seem like a fertile and pleasant field to enter. My hope is that we may find some way to remedy this situation. In addition, we must not forget our medical schools and instructors, who are molding our followers. We must especially keep in mind our own medical school at the University of North Dakota. Much encouragement should be offered to our instructors. They should have adequate salaries and equipment, pleasant working conditions, and help in procuring grant-in-aids for research projects.

There is much more to say, but I must conclude with the admonition to you and to me. I hope we all conduct ourselves during the coming year with much professional, political, and social dignity, so that we will merit the

trust and respect of that most important person in our lives, our patient. I want to thank every one of you for the high honor I have received. It is, in fact, the highest honor I have ever had and ever hope to have. I am approaching this office with much humility but, at the same time, an equal amount of enthusiasm and confidence that with the wholehearted help and cooperation from every member of this society, we may remove a few of the stumbling blocks that are appearing on the horizon.

What a relief it is to leave the offices of the second vice-president, first vice-president, and president-elect. Something should be done to keep these offices busy. Let me suggest that the office of chairman of important committees be routinely filled by the second vice-president, the first vice-president, and the president-elect. Any doctor entering the presidential chair should have the qualifications and experience of these important positions.

In conclusion, I am taking the liberty of reading to you a petition which is typed on a placard in my examining room. I see this many times a day, and it keeps me ever mindful of my duty and obligation to 2 most important people in my life—the Great Physician and my patient.

*Scourge me, Lord, with whip of steel
If I should labor and fail to feel
Darkness that is the blindman's share
Pain that the cripple has to bear.
Keep my pity so keen a blade
Its double edge upon me laid
Lest I minister and keep back,
Something that might fulfill the lack
Of a body born from the hand of death
Of a heart that lives on a body's breath.
Grant me skill and a steady hand.
The hour-glass and its moving hand
Challenge us who are pledged to give
Flesh and spirit their chance to live.
So little time—so great the task.
Thy understanding is all we ask.*

CHAFFEE

North Dakota State Medical Association Roster—1960

MEMBERSHIP BY DISTRICTS

First District

Amundson, Blaine F.	Dakota Clinic, Fargo
Armstrong, William B.	Dakota Clinic, Fargo
Bacheller, Stephen C.	Enderlin
Bakke, Hans	Lisbon
Barnard, Donald M.	Fargo Clinic, Fargo
Beithon, Elmer J.	Red River Valley Clinic, Wahpeton
Beithon, Paul J.	Red River Valley Clinic, Wahpeton
Beltz, Melvin E.	Wahpeton Clinic, Wahpeton
Borland, Verl G.	Fargo Clinic, Fargo
Burt, Arthur C.	405 Black Bldg., Fargo
Christoferson, Lee A.	702 1st Ave. S., Fargo
Christu, Chris M.	Fargo Clinic, Fargo
Corbus, Budd C.	314 Black Bldg., Fargo
Crim, Eleanor M.	1701 13th St. S., Fargo
Darner, Charles B.	Fargo Clinic, Fargo
Darrow, Kent E.	Dakota Clinic, Fargo
DeCesare, Francis A.	Dakota Clinic, Fargo
Dillard, James R.	311 Black Bldg., Fargo
Dodds, G. Alfred	Fargo Clinic, Fargo
Donat, T. L.	Dakota Clinic, Fargo
Engstrom, Perry H.	Red River Valley Clinic, Wahpeton

Fercho, Calvin K.	812 Black Bldg., Fargo
Fortney, Arthur C.	Fargo Clinic, Fargo
Foster, George C.	15 Broadway, Fargo
Geib, Marvin J.	702 1st Ave. S., Fargo
Gillam, John S.	Fargo Clinic, Fargo
Goff, John R.	Fargo Clinic, Fargo
Goltz, Neill F.	Fargo Clinic, Fargo
Gronvold, Frederick O.	910 Broadway, Fargo
Gustafson, Maynard B.	702 1st Ave. S., Fargo
Hall, G. Howard	Fargo Clinic, Fargo
Haugrud, Earl M.	304 Black Bldg., Fargo
Hawn, Hugh W.	624 Gate City Bldg., Fargo
Heilmann, Charles O.	Fargo Clinic, Fargo
Houghton, James F.	Dakota Clinic, Fargo
Hunter, C. M.	608 Black Bldg., Fargo
Hunter, G. Wilson	Fargo Clinic, Fargo
Irvine, Vincent S.	Lidgerwood
Ivers, George U.	424 de Lendrecie Bldg., Fargo
Jaehning, David G.	Red River Valley Clinic, Wahpeton
Klein, Alan L.	110-112 Gate City Bldg., Fargo
Kolner, Edward (Armed Forces)	Enderlin
Kulland, Roy Emanuel	102 Sheyemie St., West Fargo

Lancaster, W. E. G. Fargo Clinic, Fargo
 Landa, Marshall Dakota Clinic, Fargo
 Larson, G. Arthur 812 Black Bldg., Fargo
 Lawrence, Donald H. 306 Black Bldg., Fargo
 LeBien, Wayne E. Fargo Clinic, Fargo
 LeMar, John D. Fargo Clinic, Fargo
 Lewis, A. K. Lisbon
 Lewis, T. H. 302 Black Bldg., Fargo
 Lindsay, Douglas T. Fargo Clinic, Fargo
 Long, William H. Dakota Clinic, Fargo
 Lytle, Francis T. Fargo Clinic, Fargo
 Macaulay, Warren L. Fargo Clinic, Fargo
 Magill, Gordon B. 403 Black Bldg., Fargo
 Magness, John W. Dakota Clinic, Fargo
 Mazur, Bernard A. Dakota Clinic, Fargo
 Melton, Frank M. Dakota Clinic, Fargo
 Miller, Herbert H. 509½ Dakota Ave., Wahpeton
 Murray, James B. Dakota Clinic, Fargo
 Nellerroe, C. W. Fargo Clinic, Fargo
 Norum, Henry A. Fargo Clinic, Fargo
 Poindexter, M. H., Jr. Fargo Clinic, Fargo
 Poole, Ernest E. Lidgerwood
 Pray, Laurence G. Fargo Clinic, Fargo
 Rogers, Robert G. Dakota Clinic, Fargo
 Schleinitz, Fritz B. Hankinson
 Schneider, Joseph F. 114 Broadway, Fargo
 Sedlak, Oliver A. Dakota Clinic, Fargo
 Sessums, John V., Jr. Dakota Clinic, Fargo
 Shook, Lester D. Fargo Clinic, Fargo
 Spier, J. J. 1345 N. 5th St., Fargo
 Stafne, William A. Fargo Clinic, Fargo
 Story, Robert D. Fargo Clinic, Fargo
 Swanson, Joel C. 407 Black Bldg., Fargo
 Thompson, George R. Fargo Clinic, Fargo
 Traynor, Mack V. Fargo Clinic, Fargo
 Triggs, Perry O. Fargo Clinic, Fargo
 Ulmer, Robert J. Dakota Clinic, Fargo
 Veitch, Abner Lisbon
 Wall, Wendell H. Wahpeton Clinic, Wahpeton
 Wasemiller, E. R. Wahpeton Clinic, Wahpeton
 Webster, William O. Fargo Clinic, Fargo
 Weible, Ralph D. Dakota Clinic, Fargo
 Wiltse, Glenn L. Wahpeton Clinic, Wahpeton
 Wold, Lester E. Fargo Clinic, Fargo
 Zauner, Richard J. 708 Black Bldg., Fargo

Second District

Cook, Stuart J. Rolette
 Corbett, Conner A. Lake Region Clinic, Devils Lake
 Coultrip, R. L., Jr. McVillie
 Engesather, J. A. D. Lakota
 Eyelands, Jon V. Rolla
 Fawcett, John C. Lake Region Clinic, Devils Lake
 Fawcett, Robert M. Lake Region Clinic, Devils Lake
 Fox, William R. Johnson Clinic, Rugby
 Gilchrist, Milton R. 513 Buttonwood St.,
 Anaheim, Calif.
 Corrie, William A. Maddock
 Hiltz, George H. Cando
 Johnson, C. G. Johnson Clinic, Rugby
 Keller, E. T. Johnson Clinic, Rugby
 Lazareck, I. L. 411 4th Ave., Devils Lake
 Longmire, L. T. 411 4th Ave., Devils Lake
 McBane, Robert D. Lake Region Clinic, Devils Lake
 MacDonald, John A. Cando
 Mahoney, James H. 411 4th Ave., Devils Lake
 Munro, J. A. Rolla
 Owens, Clarence G. New Rockford
 Palmer, D. W. Cando

Pine, Louis F. Lake Region Clinic, Devils Lake
 Schwinghamer, E. J. New Rockford
 Seibel, Glenn W. New Rockford
 Sihler, William F. Mann Block, Devils Lake
 Stickelberger, Josephine S. 1524 Portland Ave.,
 St. Paul 4, Minn.
 Terlecki, Jaroslaw Minnewaukan
 Toomey, Glenn W. Lake Region Clinic, Devils Lake
 Vigeland, George N. Johnson Clinic, Rugby
 Voglewede, William C. Carrington

Third District

Bakewell, William E. Grand Forks Clinic, Grand Forks
 Benson, T. Q. 1600 University Ave., Grand Forks
 Benwell, Harry D. Valley Medical Associates,
 Grand Forks
 Campbell, Duncan W. Grand Forks Clinic, Grand Forks
 Campbell, Robert D. 323 S. 6th St., Grand Forks
 Cardy, James D. U.N.D. Medical School, Grand Forks
 Clark, Rodney Grand Forks Clinic, Grand Forks
 Clayburgh, Bennie J. Grand Forks Clinic, Grand Forks
 Countryman, G. L. Grafton
 Culmer, A. E., Jr. 501 1st Natl. Bank Bldg.,
 Grand Forks
 Dailey, Walter C. Valley Medical Associates,
 Grand Forks
 Deason, Frank W. 643 Cooper Ave., Grafton
 DeLano, Robert H. Northwood
 Doss, R. Douglas 1600 University Ave., Grand Forks
 Eaton, L. P. Grafton Clinic, Grafton
 Evans, Harold W. Grand Forks Clinic, Grand Forks
 Flaten, Alfred N. Edinburg
 French, Harley E. 402 Harvard St., Grand Forks
 Frey, Welde W. Drayton
 Gaspel, Cyril J. Grafton Clinic, Grafton
 Goehl, R. O. Grand Forks Clinic, Grand Forks
 Graham, C. M. 1600 University Ave., Grand Forks
 Graham, John H. 15½ S. 3rd St., Grand Forks
 Grinnell, Ernest L. Grand Forks Clinic, Grand Forks
 Hardy, Nigel A. Minto
 Harwood, T. H. U.N.D. Medical School, Grand Forks
 Haugen, C. O. Larimore
 Haunz, Edgar A. Grand Forks Clinic, Grand Forks
 Helenbolt, Kenneth S. 1600 University Ave.,
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 Helgason, Norman M. Cavalier
 Helm, Richard K. 111 N. 5th St., Grand Forks
 Hill, Frank A. Grand Forks Clinic, Grand Forks
 Jensen, August F. 1600 University Ave., Grand Forks
 Johanson, John F. Cavalier
 Kaluzniak, Nicholas Langdon
 Keig, William P., Jr. 1600 University Ave., Grand Forks
 Landry, L. H. Walhalla
 Leigh, James A. 111 N. 5th St., Grand Forks
 Leigh, Ralph E. 111 N. 5th St., Grand Forks
 Leigh, Richard H. 1600 University Ave., Grand Forks
 Levine, Leonard Grand Forks Clinic, Grand Forks
 McLeod, John Grand Forks Clinic, Grand Forks
 Mahowald, Ralph E. 2 N. 3rd St., Grand Forks
 Mann, Hamish 1600 University Ave., Grand Forks
 Marshall, Robert A. 27 S. 3rd St., Grand Forks
 Meredith, William C. Drayton
 Moore, J. H. Grand Forks Clinic, Grand Forks
 Muus, Jacob M. McVillie
 Muus, O. Harold 502 Commercial Exchange Bldg.,
 Grand Forks
 Nelson, Wallace W. Grand Forks Clinic, Grand Forks
 Nelson, William C. Grand Forks Clinic, Grand Forks
 O'Toole, James K. Park River

Painter, Robert C.	Grand Forks Clinic, Grand Forks	Larson, Richard S.	Velva
Panek, A. F.	Milton	Leonard, Kenneth O.	Garrison Clinic, Garrison
Peake, Frances M.	204 Widlund Bldg., Grand Forks	Loeb, George L.	5407 26th Ave. S., Minneapolis 17
Peterkin, F. D.	Langdon	London, Carl B.	Northwest Clinic, Minot
Pettit, Samuel L.	Grand Forks Clinic, Grand Forks	McConville, Edward B.	Harvey
Piltingsrud, Harold R.	Park River	McCullough, William F.	Bottineau
Porter, Charles B.	Grand Forks Clinic, Grand Forks	McDougall, James R.	214 S. Main St., Minot
Powers, William T.	Valley Medical Associates, Grand Forks	Malvey, K. P.	Malvey Clinic, Bottineau
		Manzanero, F. M.	McCannel Clinic, Minot
Prochaska, Leonard J.	517 1st Natl. Bank Bldg., Grand Forks	Naegeli, Frank D.	Northwest Clinic, Minot
		Nelson, Leslie F.	410 Main, Bottineau
Ralston, Lloyd S.	Grand Forks Clinic, Grand Forks	Ohrt, Harry A.	Kenmare
Rand, Charles C.	Grafton	Olson, Burton G.	McCannel Clinic, Minot
Rrud, John E.	1st Natl. Bank Bldg., Grand Forks	Richardson, Gale R.	12 10th St. S.W., Minot
Sandmeyer, John A.	Grand Forks Clinic, Grand Forks	Rowe, Paul H.	Northwest Clinic, Minot
Silverman, Louis B.	Grand Forks Clinic, Grand Forks	Sahl, Jens, Jr.	Northwest Clinic, Minot
Tarpley, Harold I.	Valley Medical Associates, Grand Forks	Seifert, G. S.	Northwest Clinic, Minot
		Shea, Samuel E.	McCannel Clinic, Minot
Teevens, William P.	Grafton Clinic, Grafton	Sorenson, Alfred R.	Medical Arts Clinic, Minot
Thorgrimsen, C. G.	1600 University Ave., Grand Forks	Sorenson, Roger	Medical Arts Clinic, Minot
Tompkins, C. R.	1004 Hill Ave., Grafton	Towarnicky, Marvin J.	Fessenden
Witherstine, W. H.	111 N. 5th St., Grand Forks	Uthus, O. S.	21½ 2nd Ave. S.E., Minot
Woutat, P. H.	Grand Forks Clinic, Grand Forks	Vaaler, Raymond A.	Great Plains Clinic, Minot
Woytassek, L. E.	Larimore	Wall, Willard W.	Northwest Clinic, Minot
Youngs, Nelson A.	Grand Forks Clinic, Grand Forks	Wallis, Marianne	St. Joseph's Hospital, Minot
Yury, Walter E.	1004 Hill Ave., Grafton	Wilson, Herbert J.	New Town

Fourth District

Anstutz, Kenneth N.	Northwest Clinic, Minot
Ayash, John J.	123 2nd Ave. S.E., Minot
Bernudez, Enrique	Garrison Clinic, Garrison
Blatherwick, Robert	Parshall
Boyle, John T.	Garrison
Boynn, Lowell E.	Harvey Medical Center, Harvey
Boynn, P. A.	Harvey Medical Center, Harvey
Breslich, Paul J.	Northwest Clinic, Minot
Briggs, Brian E.	Great Plains Clinic, Minot
Cameron, Angus L.	Northwest Clinic, Minot
Cipolla, Victor S.	Medical Arts Clinic, Minot
Cuadrado, A. R.	Tuberculosis Sanatorium, San Haven
Devine, J. L.	Great Plains Clinic, Minot
Diduch, Alexander	Stanley
Dormont, Richard E.	Northwest Clinic, Minot
Erenfeld, F. R.	617 2nd St. N.W., Minot
Fischer, V. J.	Medical Arts Clinic, Minot
Floth, Milford G.	Stanley
Floch, John L.	Mohall
Gammell, Robert T.	Kenmare
Garrison, M. W.	Garrison Bldg., Minot
Giltner, Lloyd A.	Medical Arts Clinic, Minot
Goodman, Robert	Powers Lake
Gozum, Ekrem	123 2nd Ave. S.E., Minot
Greene, E. E.	Westhope
Halliday, David J.	Kenmare
Halverson, C. H.	1st Natl. Bank Bldg., Minot
Hammargren, August F.	Harvey Medical Center, Harvey
Hart, George M.	Northwest Clinic, Minot
Heidorn, G. H.	Great Plains Clinic, Minot
Herba, Edward J.	Mohall
Hoopes, Lorman L.	17A S. Main St., Minot
Hordinsky, B. Z.	Drake
Huntley, W. B.	Great Plains Clinic, Minot
Hurly, William C.	Medical Arts Clinic, Minot
Johnson, O. W.	Johnson Clinic, Rugby
Kaemerle, Harold K.	Childrens' Hospital, Los Angeles
Kennott, L. H.	401 S. Main St., Minot
Kohl, D. L.	123 2nd Ave. S.E., Minot
Lampert, M. T.	107 1st Natl. Bank Bldg., Minot

Fifth District

Christianson, Gunder	117 N.W. 3rd, Valley City
Goven, John W.	117 N.W. 3rd, Valley City
Jensen, Warren R.	130 Central Ave. S., Valley City
Klein, C. J.	117 N.W. 3rd, Valley City
Macdonald, Alexander C.	130 Central Ave. S., Valley City
Macdonald, Neil A.	130 Central Ave. S., Valley City
Merrett, Joseph	117 N.W. 3rd, Valley City
Van Houten, J.	105 Main St. W., Valley City

Sixth District

Anderson, F. E.	Underwood
Ameson, Charles A.	412½ Main, Bismarek
Baumgartner, Carl J.	Quain & Ramstad Clinic, Bismarek
Berg, H. Milton	Quain & Ramstad Clinic, Bismarek
Berg, Roger M.	Quain & Ramstad Clinic, Bismarek
Bertheau, Herman J.	Linton
Blumenthal, P. L.	107 1st Ave. N.W., Mandan
Bodenstab, William H.	520 Mandan St., Bismarek
Boerth, Edwin H.	Quain & Ramstad Clinic, Bismarek
Brink, Norvel O.	Quain & Ramstad Clinic, Bismarek
Buckingham, T. W.	405½ Broadway, Bismarek
Cartwright, John T.	Missouri Valley Clinic, Bismarek
Clary, Joseph W.	Missouri Valley Clinic, Bismarek
Cniskis, Adolfs A.	Elgin
Dahl, Phillip O.	Missouri Valley Clinic, Bismarek
Diven, Willbur L.	402½ Main, Bismarek
Dunnigan, R. J.	Capital City Clinic, Bismarek
Eriksen, Johan A.	Quain & Ramstad Clinic, Bismarek
Evangelista, Teofilo	McClusky
Ewert, Arthur O.	Benlah
Fisher, Albert M.	922 8th St., Bismarek
Freise, Paul W.	Quain & Ramstad Clinic, Bismarek
Froeschle, R. P.	Hazen
Gache, Otto C.	New Salem
Garrett, W. G.	Missouri Valley Clinic, Bismarek
Giard, Bernard A.	Mandan
Goodman, Edward	Napoleon
Goughmour, Myron W.	Capital City Clinic, Bismarek
Gregware, P. Roy	Quain & Ramstad Clinic, Bismarek
Griehenow, Frederick	905 9th St., Bismarek

Gutowski, Franz	Wishek	Larson, E. J.	DePuy-Sorkness Clinic, Jamestown
Hamilton, Charles A.	Quain & Ramstad Clinic, Bismarek	Lucy, Robert E.	DePuy-Sorkness Clinic, Jamestown
Hanson, Harris D.	Quain & Ramstad Clinic, Bismarek	Lynde, Roy	Ellendale
Heffron, Maurice M.	405½ Broadway, Bismarek	Martin, Clarence S.	Kensal
Henderson, Robert W.	Capital City Clinic, Bismarek	McFadden, Robert L.	DePuy-Sorkness Clinic, Jamestown
Hetzler, Arnold E.	104 3rd Ave. N.W., Mandan	Melzer, Simon W.	Woodworth
Iecnogle, Grover D.	State Hospital, Jamestown	Memier, H. J.	Oakes
Jacobson, M. S.	Elgin	Miles, James V., Jr.	119 2nd Ave. S.E., Jamestown
Johnson, Kenneth J.	Quain & Ramstad Clinic, Bismarek	Nierling, R. D.	DePuy-Sorkness Clinic, Jamestown
Johnson, M. J. E.	Quain & Ramstad Clinic, Bismarek	Oster, Ellis	Ellendale
Johnson, Paul L.	Quain & Ramstad Clinic, Bismarek	Palmer, John C.	State Hospital, Jamestown
Kalnins, Arnold	Washburn	Pederson, T. E.	DePuy-Sorkness Clinic, Jamestown
Kling, Robert R.	Quain & Ramstad Clinic, Bismarek	Sorkness, Joseph	DePuy-Sorkness Clinic, Jamestown
Kuplis, Haralds	Turtle Lake	Swenson, John A.	DePuy-Sorkness Clinic, Jamestown
Larson, Leonard W.	Quain & Ramstad Clinic, Bismarek	Thakor, S. J.	State Hospital, Jamestown
Levi, Wesley E.	Quain & Ramstad Clinic, Bismarek	Turner, Neville W.	LaMoure
Lindelow, O. V.	Missouri Valley Clinic, Bismarek	Van der Linde, John M.	Medical Arts Clinic, Jamestown
Lipp, George R.	405½ Broadway, Bismarek	Van Houten, Richard W.	Oakes
Lommen, M. A. K.	Capital City Clinic, Bismarek	Woodward, Robert S.	DePuy-Sorkness Clinic, Jamestown
McGee, William J.	104 Missouri Drive, Riverdale		
Montz, Charles R.	Quain & Ramstad Clinic, Bismarek		
Morton, James R.	Quain & Ramstad Clinic, Bismarek		
Nuessle, Robert F.	Quain & Ramstad Clinic, Bismarek		
Nugent, Milton E.	Quain & Ramstad Clinic, Bismarek		
Oja, Karl F.	Ashley		
Orchard, Welland J.	Linton		
Owens, Percy L.	Missouri Valley Clinic, Bismarek		
Perrin, Edwin D.	Quain & Ramstad Clinic, Bismarek		
Peters, C. H.	Quain & Ramstad Clinic, Bismarek		
Peterson, Alice H.	State Health Department, Capitol Bldg., Bismarek		
Pierce, W. B.	Quain & Ramstad Clinic, Bismarek		
Pierson, R. Warren	Quain & Ramstad Clinic, Bismarek		
Quain, Eric P.	2075 Raynor St., Salem, Ore.		
Schoregge, C. W.	Quain & Ramstad Clinic, Bismarek		
Schoregge, R. D.	Quain & Ramstad Clinic, Bismarek		
Smeenk, H. Pieter	Quain & Ramstad Clinic, Bismarek		
Smith, Cecil C.	101 Collins Ave., Mandan		
Smith, Clyde L.	Missouri Valley Clinic, Bismarek		
Spielman, George H.	305 1st Ave. N.W., Mandan		
Stangebye, T. L., Jr.	Quain & Ramstad Clinic, Bismarek		
Thompson, Arnold	Quain & Ramstad Clinic, Bismarek		
Tudor, Robert B.	Quain & Ramstad Clinic, Bismarek		
Van Drunen, H. A.	Quain & Ramstad Clinic, Bismarek		
Vinje, Edmund G.	Hazen Clinic, Hazen		
Vinje, Ralph	405 E. Broadway, Bismarek		
Vonnegut, Felix F.	Linton		
Waldschmidt, R. H.	Quain & Ramstad Clinic, Bismarek		
Waldschmidt, W. D.	Quain & Ramstad Clinic, Bismarek		
Walter, P. A. F.	Hazen Clinic, Hazen		
Weyrens, P. J.	Hebron		
Zukowsky, Anthony	Steele		

Seventh District

Beall, John A.	320 1st Ave. N., Jamestown
Bolliger, Eugene F.	Ellendale Clinic, Ellendale
Craychec, Walter A.	Oakes
Cukurs, Paul	State Hospital, Jamestown
Elsworth, John N.	DePuy-Sorkness Clinic, Jamestown
Engberg, Roger D.	DePuy-Sorkness Clinic, Jamestown
Fergusson, V. D.	Edgeley
Freeman, John	Nebraska Psychiatric Institute, Omaha
Groncwald, T. W.	State Hospital, Jamestown
Harris, T. A.	Medical Arts Clinic, Jamestown
Hieb, Edwin O.	DePuy-Sorkness Clinic, Jamestown
Hogan, Clifford W.	DePuy-Sorkness Clinic, Jamestown
Jansonius, J. W.	Medical Arts Clinic, Jamestown
Jestadt, John J.	DePuy-Sorkness Clinic, Jamestown
Klassen, Rudolph A.	LaMoure Clinic, LaMoure

Eighth District

Borrud, Chester C.	Harmon Park Clinic, Williston
Craven, John P.	411 Main St., Williston
Craven, Joseph D.	411 Main St., Williston
Ellis, Gordon E.	Harmon Park Clinic, Williston
Fennell, William L.	Crosby
Hagan, Edward J.	411 Main St., Williston
Johnson, Alan K.	Williston Clinic, Williston
Johnson, P. O. C.	Watford City
Keller, John M.	Williston Clinic, Williston
Korwin, J. J.	120 Main St., Williston
Lund, Carroll M.	Williston Clinic, Williston
McPhail, Clayton O.	Crosby
Pile, Duane F.	Crosby
Skjei, Donald E.	Williston Clinic, Williston
Strinden, Dean R.	Harmon Park Clinic, Williston
Walker, H. Charles, Jr.	411 Main St., Williston
Wright, Willard A.	Williston Clinic, Williston

Ninth District

Ahlness, Paul	Bowman
Buckingham, W. M.	Elgin
Bush, Clarence A.	Beach
Cameron, D. Murray	Hettinger
Dukart, C. R.	Dickinson Clinic, Dickinson
Dukart, R. J.	Dickinson Clinic, Dickinson
Foster, Keith G.	Rodgers-Gumper Clinic, Dickinson
Gilliland, R. F.	Dickinson Clinic, Dickinson
Gilsdorf, A. R.	Dickinson Clinic, Dickinson
Guloien, Hans E.	Dickinson Clinic, Dickinson
Gumper, A. J.	Rodgers-Gumper Clinic, Dickinson
Hanewald, Walter C.	Dickinson
Hankins, Robert E.	Mott
Hill, S. W.	Regent
Hilts, Joseph A.	Hettinger
Larsen, Harlan C.	Rodgers-Gumper Clinic, Dickinson
Maercklein, Otto C.	Mott
Ordahl, Norman B.	Rodgers-Gumper Clinic, Dickinson
Raasch, Richard F.	Dickinson Clinic, Dickinson
Reichert, D. J.	24 W. Villard, Dickinson
Reichert, H. L.	24 W. Villard, Dickinson
Rodgers, R. W. R.	Rodgers-Gumper Clinic, Dickinson
Schumacher, William A.	12102 Silver Fox Rd., Los Alamitos, Calif.
Skwarok, Walter S.	Hebron
Slominski, Henry	Richardton
Smith, Oscar M.	P.O. Box 1188, Dickinson
Spear, Albert E.	610 1st Ave. W., Dickinson
Thom, Robert C.	Bowman

Tenth District

Dekker, Omar D.
LaFleur, Harold A.
Little, James M.
Little, Roy C.
McLean, Robert W.

Finley
Mayville
Mayville
Mayville
Hillsboro

Mergens, Daniel N.
Rosenberg, Mervin
Vandergon, K. G.
Wakefield, Kenneth M.
Waydeman, H. B.

Hillsboro
Northwood
Portland
Cooperstown
Hunter

TRANSACTIONS, FOURTEENTH ANNUAL MEETING OF THE WOMAN'S AUXILIARY TO THE NORTH DAKOTA STATE MEDICAL ASSOCIATION Grand Forks, April 30, May 1, 2, and 3, 1960

The fourteenth annual meeting of the Woman's Auxiliary to the North Dakota State Medical Association was held at the Grand Forks YWCA, Monday, May 2, 1960 at 9:30 A.M. The meeting was formally opened by Mrs. J. M. Van der Linde, president.

As Mrs. R. W. McLean, president-elect, had not yet arrived, the pledge of loyalty was given by Mrs. V. J. Fischer and repeated in unison by the members present.

Mrs. L. T. Longmire, first vice-president, gave the invocation.

A welcome to the convention was extended by Mrs. William P. Keig, Grand Forks. The response was given by Mrs. K. G. Vandergon, Portland.

The roll was called by the secretary, and the following were present:

State officers: Mrs. J. M. Van der Linde, president; Mrs. R. W. McLean, president-elect; Mrs. L. T. Longmire, first vice-president; Mrs. J. W. Jansonius, second vice-president; Mrs. Clyde Smith, recording secretary; Mrs. R. L. McFadden, corresponding secretary; and Mrs. Carl Baumgartner, treasurer.

Committee chairmen: Mrs. V. J. Fischer, nominating committee; Mrs. M. M. Heffron, publicity; Mrs. J. D. Gady, legislation; Mrs. G. D. Gertson, historian; Mrs. E. L. Grinnell, parliamentarian; Mrs. Henry Kernott, Jr., Bylaws; Mrs. Robert Hankins, editor; and Mrs. R. H. Waldschmidt, medical student loan fund.

District presidents: Mrs. E. A. Hannz, Grand Forks, and Mrs. H. W. Norum, Fargo.

Delegates: Mrs. O. M. DeMouilly, Bismarck; Mrs. M. E. Nugent, Bismarck; Mrs. D. W. Kohl, Minot; Mrs. M. W. Garrison, Minot; Mrs. P. Thakor, Jamestown; Mrs. C. M. Lund, Williston; and Mrs. W. Hancwald, Richardson.

Councillors: Mrs. Keith Vandergon, Portland.

Dr. J. C. Fawcett, president of the North Dakota State Medical Association, was introduced by Mrs. Van der Linde. He brought greetings from the NDSMA. He pointed out the challenge that 1960 and 1961 will bring and asked for action in the fields of public relations and legislation.

The following "in memoriam" for Mrs. Margaret T. Mulligan and Mrs. George H. Spielman was given by Mrs. J. W. Jansonius:

"We of the auxiliary are saddened by the loss of 2 of our members—Mrs. Margaret T. Mulligan, Grand Forks, and Mrs. George H. Spielman, Mandan.

"Mrs. Margaret T. Mulligan, wife of the late Dr. Thomas Mulligan, died October 17, 1959. She was born in Seaforth, Ontario, June 14, 1883, and attended Sacred Heart Academy in London, Ontario. In 1908, she married Dr. Mulligan, who practiced in Grand Forks from 1904 until his death in 1937. Mrs. Mulligan continued to live in the home built in the first year of their marriage. Their 5 children died in infancy. She was a member of St. Michael's Alter Society and a charter member of St. Mary's Alter Society. During her husband's lifetime, Mrs. Mulligan devoted much of her time visiting his patients in the hospital and often in homes on the prairie, to which she had to travel by buggy or sled.

"Mrs. George H. Spielman of Mandan died on Sep-

tember 2, 1959. She had been in failing health for the past ten years. She was a charter and founding member of the Sixth District Auxiliary and daughter of a pioneer Dakota family. She came to Flasher when she was 11 and moved to nearby Mandan in 1917, so, for over fifty years, she contributed color, spirit, and culture to these neighboring communities. Mrs. Spielman was a member of the Presbyterian church and served on the Mandan Library board for a number of years. We regret that her retirement from our auxiliary ranks some years ago deprived us of closer acquaintance with her. Dr. Spielman and 3 sons survive.

"We of the auxiliary extend our deepest sympathy to the families and offer this memoriam."

Mrs. R. W. McLean moved that we accept the minutes of the thirteenth annual meeting of the Woman's Auxiliary to the North Dakota State Medical Association as published in the October 1959 issue of THE JOURNAL-LANCET. Motion carried.

Mrs. Van der Linde stated that her report was given at the House of Delegates and would be printed in THE JOURNAL-LANCET. She then called for the reports of the state officers, which are as follows:

Treasurer's Report, Financial Statement, 1959-1960

<i>Income:</i>		
Balance on hand July 1, 1959		\$1,826.02
Dues: 319 members at \$4.00	\$1,276.00	
4 associate members at \$1.00	4.00	
Student loan contributions	1,951.00	
Essay contest (men's society)	90.00	
Convention expenses (men's society)	200.00	
Convention registration	109.00	
Sale of 1 Handbook	.50	
	\$3,630.50	3,630.50
Total receipts		\$5,456.52
<i>Disbursements:</i>		
Mrs. John Van der Linde, state president	\$ 510.08	
Mrs. Robert McLean, president-elect	120.00	
Convention expenses	260.00	
<i>News, Views and Cues</i>	271.91	
Standing committees	56.45	
Essay Winners:	90.00	
Polly DuBourt	\$50.00	
Patricia Morris	25.00	
Judy Aver	15.00	
National treasurer:		
323 members at \$1.00	323.00	
Student loan Dean Harwood	1,951.20	
Presidents pin	7.40	
AMEF	25.00	
Stationery	49.59	
Printing of Bylaws booklets	42.74	
Total expenditures:	\$3,707.40	3,707.40
Balance on hand July 1, 1960		\$1,749.12
AMEF donations (by AMEF chairman)	\$525.21	

CECILE BAUMGARTNER, State Treasurer

Membership Report, 1959-1960

The members of the Woman's Auxiliary to the North Dakota Medical Association total 324. Of this number, 312 are regular members, 7 are members at large, 4 associate members, 1 honorary member.

There are 10 organized medical districts in our state and 10 district medical auxiliaries. No new districts were organized during the past year.

At the time of the filing of this report, 27 did not renew their membership. Of last year's members, 10 have left the state, and 1 member is deceased.

MRS. R. W. McLEAN, Chairman

Program Report, 1959-1960

Throughout North Dakota, auxiliary members have worked to carry out the 1959-1960 theme "Accept Individual Responsibility for Better Community Health." Most of this work was done on an individual basis rather than as a group project. Members of the Fargo district have served the senior citizens of their community at their bimonthly meetings. In most of the districts, talks were given by auxiliary members before PTA and other groups on the dangers of plastic film in the home. Programs were presented on the care of the aging and community responsibility.

In each district, the Forand bill was discussed at one meeting, usually when our president, Mrs. J. Van der Linde, visited the group. Members of the auxiliary worked tirelessly to acquaint the public with the facts about this legislation.

In the field of recruitment, 3 districts sponsor Future Nurses Clubs, 1 has an active Committee on Paramedical Careers, and several work with the local schools on Careers Day. Individual members work with hospital auxiliaries in recruitment.

AMEF note paper sold to members and memorials, Christmas cards, and donations from district treasuries have made up our contributions to AMEF. More should and must be done for AMEF and for our State Medical Student Loan Fund in order to keep our medical schools free of government control. Auxiliary members in Fargo and Grand Forks gave dinner dances for the Student Loan Fund and reported enthusiastic response to this type of fund raising. Other district groups incorporate their contributions in their dues.

The safety slides "Car of His Own" were shown in the Richardton and Devils Lake high schools. This excellent material should be presented to every teen-age driver in the state. Minot had a driving instructor speak on "Safe Driving." Following this meeting, members participated individually in a driving check-up with the instructor.

Response to the AAPS essay contest was about the same as in past years. Six districts promoted the contest and contacted the schools in their areas. Some reported poor response, but all are willing to keep trying in the future.

Most of our North Dakota districts are in rural areas, and road and weather conditions affect attendance in all but the larger cities. For this reason, it is difficult for most of our districts to plan specific programs. Some of them have become purely social groups. In spite of this handicap, members have been carrying out national auxiliary programs in their own communities and have made their local areas aware that doctors' wives do much to promote better community health.

MRS. THOMAS LONGMIRE, Chairman

Press Report, 1959-1960

An innovation was accomplished this year by having the 4 issues of *News*, *Views*, and *Cues* printed by a commercial firm. The format and appearance improved immeasurably, and it simplified assembly. However, cost increased proportionately as to printing versus stencilling, but postage and handling decreased. This was partially accomplished by sending copies of only the first issue to all of the doctors' wives in the state and mailing the last 3 issues to only active auxiliary members, as was suggested at the last convention.

There are 3 members on the staff of *News*, *views*, and *Cues*. Mrs. C. A. Arneson, Bismarck, was in charge of mailing lists and budget. Mrs. J. H. Mahoney, Devils Lake, was managing editor in charge of committee reports, presidents' and officers' messages, editorial policy, and personal sketches. She was also in charge of proof-reading and printing. Since the printing firm was located in Devils Lake, she also took care of mailing. Mrs. R. E. Hankins, Mott, was district news editor. Her duties included contacting the district councillors, receiving and editing their copy and compiling it.

It was through the excellent cooperation of the contributors and councillors and their committees that this committee was able to function. Thank you for your assistance.

MRS. R. E. HANKINS, Substituting for
MRS. J. H. MAHONEY, Managing Editor

District News Chairman's Report, 1959-1960

News coverage from most districts was satisfactory. Jamestown and Kotana each sent news for 2 issues; First District for three issues; and Second, Third, Northwest, Sixth and Southwest were each covered in all 4 issues—October, November, February, and April. Sheyenne Valley and Traill-Steele were the only districts that sent no news.

With our new format and the very pleasant effect of the printing, we find that we have much more room for news articles than was expected. Therefore, for the following, we will be able to accept any newsworthy items. We had suggested cutting down on travel articles, but found we did not have to cut any of these because of lack of space. A breakdown of subjects covered consists of the following: meetings and auxiliary projects, 39; community service, 60; membership, 11; in memoriam, 5; illnesses, 4; progeny births, 20; achievements, 4; foreign travel, 4; state and national meetings, 11; other, 10; and miscellany, 2. So, we see that the broadest coverage was given to the most important subjects—auxiliary projects and community service.

It is again suggested that the councillors try to contact their entire membership at least once each year, either by telephone or post card. Coverage could be more complete if the entire membership could be contacted for each issue. Several of the larger districts have found this feasible by using a committee of reporters, each of whom is responsible for contacting about 10 persons whose names are divided alphabetically, thus curtailing "clinic" or "specialty" rivalries and actually bringing the groups in closer social contact. I would like to suggest that the councillors be supplied with double post cards, if the budget permits, to send to out-of-town members especially.

MRS. R. E. HANKINS, District News Editor

Publicity Report

Pursuant to a decision reached at our 1959 convention, a new chairmanship for publicity was created. In pre-

vions years, publicity was handled by a press and publicity chairman, who was also managing editor of *News, Views, and Cues*.

Upon the recommendations of both the retiring and incoming managing editors of our official publication, it was deemed practical to add a new state board member for publicity alone. Editorial duties entail sufficient work for 1 volunteer, and the editor could work more efficiently if unhampered by publicity work. The jobs actually have very little interrelationship; in fact, the deadlines for *News, Views, and Cues* and for a publicity news release often conflicted, resulting in a frustrating compromise between the 2 jobs. It was hoped also that when publicity became the sole responsibility of 1 chairman, better publicity would result. There was, incidentally, no exact parallel in our parent organization, the medical association, nor in the national auxiliary to guide us in deciding how to handle publications and publicity because both of these organizations have paid executive secretaries to handle details of both projects.

Four publicity projects were completed this year:

1. At the fall board meeting, mimeographed copies of a news release to be used for newspaper publicity in each district were distributed. This prepared copy contained mention of those participating in the board meeting, state and national priority projects, and the promotion of and details about the AAPS essay contest.

2. In April, another news release announcing the state winners in the essay contest was sent to all district presidents.

3. A survey of how much and what kind of publicity each district had procured was also made in April.

4. At convention time, a news release received from the Woman's Auxiliary to the American Medical Association was given to our newspapers. It mentioned details of the national auxiliary meeting and names of the delegates from North Dakota.

Results of the survey of district publicity revealed that most local newspapers are very cooperative in giving space to our meetings and projects. It was also apparent that the auxiliary received the most publicity in districts which have a publicity chairman or a councillor who handles both the *News, Views, and Cues* and the community publicity. Several districts had fine publicity projects which other districts might want to note and adopt, such as the TV program given by the Future Nurses Club, which was sponsored by Southwestern District; the tea which this auxiliary gives for future nurses and practical nurses at the hospital; speeches by auxiliary members at women's club meetings to explain the Forand bill, as was done in First District; or arranging for newspaper photographers to take pictures of essay contest winners receiving awards, as the Grand Forks District did. Other districts could follow Stutsman District's practice of stressing in its newspapers the work which the auxiliary does in community service projects. First District's practice of arranging for pictures of its members at work on their projects to accompany their publicity, or Northwest District's alertness which procured for them the necessary and desired newspaper interest when its members took the safe driving test.

It is recommended that every district president appoint a publicity chairman for 1960 and 1961 and that the names and addresses of the district chairmen reach the state publicity chairman no later than September 1. It also would seem desirable to procure state-wide publicity when this year's Student Loan Fund check is presented to the University of North Dakota Medical School.

Mrs. M. M. HEFFRON, Chairman

Civil Defense Report, 1959-1960

During this current auxiliary year, I have sent material pertaining to civil defense preparedness, home preparedness awards, and so forth 3 times to the individual civil defense chairmen. In my covering letters, included each time with the enclosures, I stressed the special need for wives of doctors to be so prepared. In February, I mailed out report forms to be filled and returned to me. I received 6 replies, the answers from which are compiled on the national form.

I received correspondence from Mrs. Woodward and Mrs. Dunlevy and wrote to the latter concerning tornado protection preparedness in this area.

In February, I also advised the chairmen of the two-week National Security Seminar planned in Fargo during March, and offered to secure tickets for interested members. I received no responses but attended the lectures myself and felt most fortunate to have had the opportunity to do so as they were interesting and vital.

MRS. R. D. STORY, Chairman

Safety Report, 1959-1960

Two of the district auxiliaries have reported meetings this year at which safety programs were sponsored. The First District showed a film by Bell Telephone Company called "Charlie's Haunts." The Fourth District invited a teacher of driver education to speak at one of their meetings. During a two-week period following this program, individual driving analyses were given to members of the auxiliary. Members participating in these tests found worthwhile points for improvement for better and safer driving. The publicity chairman saw that this particular activity was called to the attention of the public. A notice appeared in the local paper headed "Northwest District Medical Auxiliary Members Given Driving Analyses."

Some of the districts worked on safety activities through the Red Cross, hospital auxiliaries, and PTA's. A member of the Southwest district used the slides and script "A Car of His Own" for a PTA program. There have been individual talks given on the misuse of plastic film.

The nature of the safety program recommended by the AMA lends itself readily to numerous community organizations. It should continue to be a healthy project bringing longer and happier lives to all of us.

MRS. LORMAN L. HOOPES, Chairman

Legislation Report, 1959-1960

The Forand bill has, in large measure, been responsible for the active interest our auxiliary members have taken in medical legislation. As far back as 1957, the Forand bill and the varied and vital reasons why it should not be passed were reported by our state legislation chairman. At that time, our attitude was one of watchful waiting.

This year, upon the request of the executive secretary of the North Dakota State Medical Association, our district auxiliaries forwarded copies of resolutions opposing the Forand bill to our North Dakota congressmen and senators as well as to the chairman of the House Ways and Means Committee. These resolutions had been discussed and prepared by our district legislation committees.

Upon the request of the chairman of legislation of the North Dakota State Medical Association, they also wrote individually to President Eisenhower, Vice-President Nixon, and Secretary Flemming congratulating them on the stand they have taken and to Senator Byrd, Head of

the Finance Committee, asking that no appropriation be made for such legislation.

An appeal requesting our members to cooperate with the North Dakota State Medical Association concerning the Forand bill was published in our *News, Views, and Cues*. The response to this appeal was immediate and gratifying. Few members, if any, failed to write or wire our senators and congressmen opposing the Forand bill.

From reports received, it can be said that the members of the Woman's Auxiliary to the North Dakota State Medical Association are now participating in medical legislation as well as showing an active interest.

In November 1959, I attended the legislative sessions of the North Central Medical Conference in Minneapolis. In January 1960, our president-elect represented me at a conference on legislation of our state and district medical societies in Fargo. At the conferences, the Forand bill and ways of combating it were the main topics of discussion. On both occasions, it was pointed out that a poor climate leaning toward socialism exists in our country and better public relations between the medical profession and the public was advised. It was stressed that, above all, we must never be complacent about medical legislation.

MRS. JAMES D. CARDY, Chairman

Paramedical Careers

Reports have not been received from all our districts; therefore, this report is not complete. However, it appears that with the exception of a very few districts, there was a lack of interest. Considerable work and research was done last year by the chairman, Mrs. John Young, and it is regrettable that the results were not put to better use this year.

Walpeton has had a very active recruiting program, but details were not included in that district's reports.

In Stutsman County, Trinity Hospital Auxiliary gave 1 scholarship to a nursing student. One Future Nurse Club has been organized in Jamestown.

Northwest District made a contribution to the Student Loan Fund, and it distributed copies of "Report on Scholarships" to 20 high schools.

Southwest District had a very active year and received much cooperation from various organizations such as study clubs, directors of hospitals, and the Practical Nursing School.

Mott Mediacorps offered 1 scholarship of \$50, and Southwest District organized 2 tours to the District Health Unit at Dickinson, to 2 hospitals in Bismarck, to the Crippled Children's Hospital, to the State Hospital, and to Jamestown College at Jamestown. It also showed films and engaged speakers to stimulate interest. It held a tea in May, at which time the practical nurses at St. Joseph's Hospital received their pins and the members of the Future Nurses Club received award pins.

The foregoing information is all that was received by me from the districts in regard to their activities. It is hoped that all districts will work more in the area of Paramedical Careers next year.

MRS. NEVILLE TURNER, Chairman

Bylaws chairman, Mrs. Henry Kermott, Jr., announced that the revised copies of the Bylaws were ready and would be distributed at the close of the morning meeting.

American Medical Education Foundation Report

As of May 15, 1960, \$525.21 has been contributed for the American Medical Education Foundation. Of this total, \$128 was realized from the sale of stationery, \$25

from state contributions, and \$43.46 from the remainder of the Christmas card project of 1959. The remainder was obtained through private donations. Contributions from the sale of stationery were received from first, second, sixth, and seventh districts. The plan has been different this year, and the funds already contributed have been sent to the Chicago office.

I think we should continue a search for a project or have each district pledge \$2 per member.

I appreciate greatly the support given me this year from all the district AMEF chairmen.

MRS. J. W. JANSONIUS, Chairman

AAPS Essay Contest Report

Four districts sent 11 essays to be judged at the state level in the 1960 AAPS essay contest. At least 3 other districts contacted schools but were unable to interest students in entering the contest. All of the district chairmen are to be commended for their efforts. The contest is being recognized more and more each year, but we must increase our efforts to reach the students of every high school in the state.

The committee of state judges consisted of Dr. R. B. Tudor, Bismarck, chairman; Dr. George W. Starcher, president of the University of North Dakota; and Mr. Carl Smith, superintendent of the North Dakota School for the Deaf at Devils Lake.

The North Dakota State Medical Association contributed \$90 in prize money. The first place prize of \$50 went to Polly Ann DuBourt, a junior at St. James High School in Grand Forks; second place prize of \$25 was won by Patricia Anne Morris, a senior at St. Mary's Academy in Devils Lake; and third place prize of \$15 was taken by Judy Aver, a junior at St. James High School in Grand Forks.

Other entries submitted to the contest were written by La Vonn Boehm, Mott Lincoln High School, Mott; Ranae Belford, St. Mary's Academy, Devils Lake; Ronald Edgar Rietz, Central High School, Devils Lake; Marie Jane Wehri, St. Mary's High School, Richardton; Lorelee Nadine Mayer, St. Mary's High School, Richardton; Diane Goser, Sacred Heart Academy, Minot; Heine Mosbrucker, Sacred Heart Academy, Minot; and De Lylia Nygard, Sacred Heart Academy, Minot.

MRS. THOMAS LONGMIRE, Chairman

Historians Report 1959-1960

The thirteenth annual meeting of the Woman's Auxiliary to the North Dakota State Medical Association was held at the Apple Creek Country Club on Monday, May 4, 1959. The pre-convention board meeting was held May 3 at the Quain & Ramstad Clinic Building. Registrations opened at 12 noon, May 3 in lobby of the Prince Hotel, Bismarck.

Official minutes are recorded in the October 1959 issue of THE JOURNAL-LANCET. Convention program and all minutes and reports are filed in the archives. The fall board meeting was held in Jamestown on September 23 and was presided over by Mrs. J. M. Van der Linde, the new president, with 26 members present.

Mrs. J. M. Van der Linde reported on the June 1959 national convention in Atlantic City's Haddon Hall Hotel. The annual conference of state presidents, presidents-elect, and national officers, which was held October 1959 in Chicago's Drake Hotel, was reported by Mrs. R. W. McLean, president-elect and was published in the October and November issues of *News, Views, and Cues*.

Honored guest speakers at state convention were Mrs.

William Mackersie, Detroit, North Central regional vice-president of the auxiliary to the AMA. Dr. O. A. Sedlak, president of the North Dakota State Medical Association; and Dr. J. C. Fawcett, president-elect of the association.

In memorial policy was established "to recognize on a state level through their past association in the organization to have the memorial read at state convention in the year they are deceased." Motion was made by Mrs. R. D. Nierling and carried. Presidential appointee is to give or send what she received to the historian for the scrapbook archives.

Registration at state convention numbered 101 members. Of the total number of auxiliary members, 324 are regulars, 4 are associates, 7 are members at large, and 1 is honorary.

Achievements on national and state levels are quoted as follows from *News, Views, and Cues*:

"Mrs. J. M. Van der Linde received a citation from the American Heart Association for her work last year.

"Mrs. R. W. Gilliland, Dickinson, is vice-president of the North Dakota Society for Crippled Children and Adults, Inc.

"Mrs. A. P. Nachtwey, Dickinson, was appointed by Governor John E. Davis to serve a five-year term on the State Merit Council.

"Award of Merit was given to the North Dakota State Auxiliary for being 1 of 10 states based on high per capita contributions to national AMIEF.

"At the national convention, the invitation to *Today's Health* subscription project honor luncheon was in recognition of the fact that North Dakota had reached the goal of 1 of 19 winning states. The honor was accepted by Mrs. Van der Linde."

Resolutions passed by the House of Delegates expressed great appreciation and thanks for the many projects and achievements . . . of the Woman's Auxiliary to the North Dakota State Medical Association. Copy of resolutions appeared in October 1959 issue of *News, Views, and Cues*.

CLARA D. GERTSON, Historian

1959 Convention Program

SUNDAY, MAY 3, 1959

- 12:00 Noon Registration in Prince Hotel, Bismarck, with Mrs. R. F. Nuessle, chairman, presiding.
- 2:00 p.m. Finance committee meeting at Quinn & Ramstad Clinic Building, with Mrs. R. H. Waldschmidt, chairman, presiding.
- 3:00 p.m. Student loan fund committee meeting, with Mrs. B. A. Mazur, chairman, presiding.
- 4:00 p.m. Pre-convention board meeting, with Mrs. V. J. Fischer, president, presiding.
- 7:00 p.m. Informal mixer and buffet Supper at Municipal Country Club.

MONDAY, MAY 4, 1959

- 8:30 a.m. Past president's breakfast, with Mrs. C. A. Arneson, chairman, presiding.
- 9:00 a.m. Registration in lobby of Prince Hotel.
- 9:00 a.m. Opening session at Apple Creek Country Club, with Mrs. V. J. Fischer presiding.
- Pledge of loyalty: Led by Mrs. J. M. Van der Linde, president-elect.
- Invocation: Mrs. R. W. Gilliland, first vice-president.
- Welcome: Mrs. P. O. Dahl, president-elect of the sixth district.
- Response: Mrs. R. L. McFadden, Jamestown.
- Greetings: Dr. O. A. Sedlak, president, NDSMA.
- In Memoriam: Mrs. R. W. McLean, second vice-president.
- Reports of state officers, chairmen and district presidents.
- 12:30 p.m. Luncheon at Apple Creek Country Club, with Mrs. J. M. Van der Linde, president-elect, presiding. Mrs. P. O. Dahl introduced honored guests. Guest speaker was Dr. J. C. Fawcett, president-elect, NDSMA.
- 2:30 p.m. Second business session at Apple Creek Country Club, with Mrs. V. J. Fischer, president, presiding. Unfinished business, new business, and election of officers took place.

6:30 p.m. Informal banquet at Bismarck Municipal Country Club, with Mrs. R. D. Schoregge, sixth district president, presiding. Honored Guest and Speaker was Mrs. William Mackersie, Detroit, regional vice-president of the auxiliary to the AMA.

TUESDAY, MAY 5, 1959

- 10:00 a.m. Brunch in the Princess Room of the Prince Hotel, with Mrs. R. H. Waldschmidt, convention chairman, presiding. The program included entertainment, installation of officers, and the post-convention board meeting.

Officers and Chairmen of Standing Committees, 1959-1960

State Officers

- President Mrs. J. M. Van der Linde, 1016 4th Ave. N.E., Jamestown
- President-elect Mrs. R. W. McLean, Hillsboro
- First vice-president Mrs. L. T. Longmire, 810 6th St., Devils Lake
- Second vice-president Mrs. J. W. Jansonius, 609 4th Ave. S.E., Jamestown
- Recording secretary Mrs. Clyde Smith, 622 Raymond St., Bismarck
- Treasurer Mrs. Carl Baumgartner, 615 N. Washington, Bismarck
- Corresponding secretary Mrs. R. L. McFadden, 910 3rd Ave. N.W., Jamestown

State Committee Chairmen

- Organization Mrs. R. W. McLean, Hillsboro
- Program Mrs. L. T. Longmire, 810 6th St., Devils Lake
- Civil defense Mrs. Robert Story, 1315 S. 9th St., Fargo
- Nominating Mrs. V. J. Fischer, 303 8th Ave. S.E., Minot
- Publicity Mrs. M. M. Heffron, 320 Ave. B West, Bismarck
- Legislation Mrs. J. D. Cardy, 1110 Reeves Drive, Grand Forks
- Bulletin Mrs. A. G. Sathe, 716 15th St. W., Williston
- Historian Mrs. G. D. Gertson, 511 S. 5th St., Grand Forks
- AMIEF Mrs. J. W. Jansonius, 609 4th Ave. S.E., Jamestown
- Parliamentarian Mrs. E. L. Grinnell, 1207 Lincoln Drive, Grand Forks
- Mental health Mrs. Keith Foster, 228 2nd Ave. E., Dickinson
- Paramedical careers Mrs. Neville Turner, LaMoure
- Bylaws Mrs. H. L. Kermott, Jr., 207 7th Ave. S.E., Minot
- Safety Mrs. L. L. Hoopes, 118 9th Ave. S.E., Minot
- AAPS Mrs. L. T. Longmire, 810 6th St., Devils Lake
- Community service Mrs. S. E. Shea, 808 1st St. S.E., Minot
- Official publications Managing editor, Mrs. J. H. Mahoney, Devils Lake; business and circulation manager, Mrs. Charles Arneson, Bismarck; and co-editor, Mrs. Robert Hankins, Mott
- Finance committee Mrs. Henry Kermott, chairman, 207 7th Ave. S.E., Minot; Mrs. E. J. Larson 321 2nd Ave. S.E., Jamestown; Mrs. Carl Baumgartner, 615 N. Washington, Bismarck; Mrs. W. L. Macaulay, 1410 S. 9th St., Fargo; and Mrs. L. E. Wold, 1708 S. 9th, Fargo.
- Medical student loan fund Mrs. R. H. Waldschmidt, chairman, 600 N. Washington, Bismarck; Mrs. J. A. Sandmeyer, 1005 Lanark, Grand Forks; Mrs. G. R. Richardson, 12 10th St. S.W., Minot; Mrs. J. M. Keller, 910 4th Ave. E., Williston; and Mrs. R. D. Nierling, 415 9th St. S.E., Jamestown.

District Presidents, 1959-1960

- First District Mrs. H. A. Norum, 1533 S. 6th St., Fargo
- Second District Mrs. J. A. Terlecki, Minnevakan
- Third District Mrs. E. A. Hanz, 1029 Lincoln Drive, Grand Forks
- Fourth District Mrs. L. Hoopes, 118 9th Ave. S.E., Minot
- Fifth District Mrs. N. A. MacDonald, 711 5th Ave. N.W., Valley City (members-at-large)
- Sixth District Mrs. Philip Dahl, 1111 S. Highland Acres Road, Bismarck
- Seventh District Mrs. J. W. Jansonius, 608 4th Ave. S.E., Jamestown
- Eighth District Mrs. John Keller, 910 4th Ave. E., Williston
- Ninth District Mrs. R. F. Raasch, Dickinson
- Tenth District Mrs. D. N. Mergens, Hillsboro

District Councillors, 1959-1960

- First District Mrs. J. H. Bond, 516 N. 13th St., Fargo
- Second District Mrs. L. T. Longmire, 810 6th St., Devils Lake
- Third District Mrs. P. H. Wontat, 1205 Lincoln Drive, Grand Forks
- Fourth District Mrs. V. J. Fischer, 303 8th Ave. S.E., Minot
- Sixth District Mrs. M. M. Heffron, 320 Ave. B West, Bismarck
- Seventh District Mrs. G. H. Holt, 214 2nd Ave. N.W., Jamestown
- Ninth District Mrs. L. Reichert, Dickinson
- Tenth District Mrs. K. Vandergon, Portland

Mrs. E. L. Grinnell, parliamentarian, read from the Bylaws the qualifications of voting delegates.

Mrs. H. L. Kermott, Jr. Finance Committee chairman, presented the proposed budget for 1960-1961. She requested Mrs. J. D. Cardy to read a letter from the president of the auxiliary to the Student American Medical Association asking for funds to send a delegate to the SAMA national convention. Mrs. Kermott stated that the sum of \$90 had been allowed in the budget for this purpose, subject to the approval of the convention.

Community Service Report, 1959-1960

A review of the community services of the auxiliaries reveals a very commendable pattern of activity in practically every phase of community endeavor, especially those relating to health and health education. Auxiliaries as organizations and members as individuals have served in many volunteer capacities, providing leadership and valuable assistance in community work. Members have rendered help with the Governor's Survey of Aging and provided recreation for Senior Citizen's Clubs. Hospital auxiliary services have been an outstanding example of volunteer work in which physicians' wives have participated. The long list of community services includes fund raising campaigns, PTA, Red Cross, Girl Scouts, Multiple Sclerosis Society, mental health, civil defense, community chest, and many others.

The AAPS essay contest has been sponsored for the third year by our North Dakota auxiliaries, and high school students have become increasingly interested.

A gratifying summary of community Services depicts the medical auxiliaries as tremendous contributors in time, effort, and leadership in all phases of community life. North Dakota can be justly proud of its auxiliary members.

MRS. SAMUEL E. SHEA, Chairman

Medical Student Loan Fund Report

Devils Lake, 16 members	\$ 64.00
First District, 70 members	300.00
Grand Forks, 52 members	420.20
Kotana, 18 members	100.00
Northwest District, 38 members	300.00
Sixth District, 62 members	600.00
(Each member of the group pays \$10 voluntarily with her dues)	
Southwest District, 25 members	50.00
Stutsman, 22 members	110.00
Traill-Steele, 9 members	7.00
Total	\$1,951.20

For the year of 1959, our annual gift was \$1,863.55 plus interest and repayments, making it possible to lend \$4,950. Loans were made to 10 students, 9 of which were for \$500 and 1 for \$450. As of December 31, 1959, the balance in the fund was \$250.46.

MARGARET WALDSCHMIDT, Chairman

Nominating Committee Report

Proposed slate for 1960-1961

President:

Mrs. R. L. McLean, Hillsboro, tenth District

President-elect:

Mrs. L. T. Longmire, Devils Lake, Second District

First vice-president:

Mrs. J. W. Jansonius, Jamestown, Seventh District

Second vice-president:

Mrs. R. H. Waldschmidt, Bismarck, Sixth District

Secretary:

Mrs. Clyde Smith, Bismarck, Sixth District

Treasurer:

Mrs. Carl Baumgartner, Bismarck, Sixth District

MRS. V. J. FISCHER, Chairman

MRS. J. H. MAHONEY

MRS. E. A. HAUNZ

MRS. O. A. SEDLAK, alternate

for Mrs. J. H. BOND

MRS. E. J. LARSON

Mrs. Van der Linde asked for nominations from the floor for each of the above offices. Since there were none, the secretary was asked to cast a unanimous ballot for the slate of officers.

Motion was made by Mrs. Longmire that the standing committee reports be accepted. Motion was seconded and carried.

Resolution Report

1. *Be it resolved* that this convention of the Woman's Auxiliary to the North Dakota State Medical Association extend to Mrs. John Van der Linde its sincere thanks and appreciation for the service which she has rendered to that group in the past year.

2. *Be it resolved* that the Woman's Auxiliary to the North Dakota State Medical Association express its appreciation and thanks to the city of Grand Forks; to the Third District Medical Society; to Mrs. James D. Cardy, convention chairman; to managers and staffs of hotels and motels; to members of the press, radio, and television; to Mr. Lyle Limond and his staff; to Dr. John Fawcett, president of the North Dakota State Medical Association; to Mrs. Stephen Bacheller, North Central regional vice-president of the Woman's Auxiliary to the American Medical Association; and to all other persons and groups who have contributed to the success of the convention.

3. *Be it resolved* that the Woman's Auxiliary to the North Dakota State Medical Association express appreciation for the help and cooperation from all persons, organizations, and agencies who contributed to the success of its program during the past year.

MRS. THOMAS LONGMIRE, Chairman

Mrs. Longmire moved that the resolution report be adopted. Motion was seconded and carried.

The following district reports were then given:

Auxiliary President's Report—First District

The First District Medical Auxiliary had 70 members this year, the largest membership since the organization of the group.

Four luncheon meetings were held this year, and attendance averaged approximately 45 members. The first meeting was held October 30, 1959, in the Tree Top Room at the Frederick Martin Hotel in Moorhead. A safety movie, "Charlie's Haunts," was shown. The second meeting on November 30 was a social meeting following the usual business meeting at the Fargo Country Club. Mrs. Stephen Bacheller gave a report on the national auxiliary meeting. The third luncheon meeting was held at the Gardner Hotel. Dr. Gertrude Donat gave a most interesting talk on mental health. Mrs. Van der Linde was our honored guest and speaker at our fourth meeting on March 29 in the Frederick Martin Hotel. Mrs. McLean was also a guest at this meeting. The election of officers took place.

In October 1959, the board members met with a group of auxiliary members and prospective members in Wahpeton in order to derive a better understanding of the purposes of an auxiliary. We hope that we will be able to work more closely with all of the committees in this district and that we may benefit from them and they from us.

Again we served the senior citizens a dessert luncheon twice a month at the YWCA. Three members cooperate on this project each time. Christmas gifts were collected and sent through the newly organized mental health organization in Fargo to the Jamestown Hospital.

February 29, 1960, a benefit dinner dance "The Silver Skates Ball" was held in the Top of the Mart at the

Frederick Hotel. Mrs. Calvin Fercho and Mrs. Richard Zanner were co-chairmen of this successful evening. One hundred tickets were sold at \$15 a couple. Proceeds were sent to the state committee. We donated \$100 to AMEF and \$300 to the Student Loan Fund.

Mrs. L. G. Pray, chairman of the Legislative Committee, contacted many organizations in regard to the Forand bill. As a result, many of these organizations passed resolutions against the bill, and individual members wrote to their congressmen expressing their opinions of the bill.

Our Committee on Paramedical Careers has worked on an informal basis this year. Mrs. Howard Hall, acting chairman, and her committee transported interested students on Career Day to the hospitals to see the x-ray departments, laboratories, and so forth. The Wahpeton members carried out an extensive recruitment program, which included students from many schools in the area.

A large percentage of our auxiliary has been active in other community projects, including The Board of Education, the Committee on Mental Health, PTA, Junior Service League, and YWCA. Numerous hours of volunteer work have been given to these organizations, as well as to the Fargo-Moorhead Symphony Orchestra, Fargo-Moorhead Opportunity School, and the United Fund. This district, as so many undoubtedly are, is organized to such an extent that we have found it is much more successful to put our best efforts on a few projects, such as our benefit dance, and then give individual help to the committees and organizations, which are already active, when it is needed.

I wish to thank all of the officers and chairmen for making my position of president a pleasure rather than a task.

MRS. HENRY A. NORUM, President

Auxiliary President's Report—Second District

The auxiliary of the Devils Lake Medical Society held 6 meetings during the 1959-1960 year. The auxiliary has 16 members.

Because most of our members live in different towns, we exist primarily as a social group; however, individually our members are very active in community organizations, such as Red Cross, PTA, Girl Scouts, Future Home Makers of America, and many others.

Mrs. D. Palmer of Cando directs a local chorus group, which gives the community the opportunity to enjoy good music.

Mrs. Thomas Longmire of Devils Lake organized a very successful campaign against the Forand bill.

We sponsored the essay contest, which was financed by the Devils Lake Medical Society.

A contribution of \$64 was made to the Student Loan Fund.

The sale of stationery for the benefit of the American Medical Education Foundation realized \$28.

MRS. JAROSLAW TERLECKI, President

Auxiliary President's Report—Third District

The Grand Forks District Medical Auxiliary, which currently has 52 members, had 4 dinner meetings during the year 1959-1960. The social chairmen made each meeting a festive occasion with decorations appropriate for the season. Miss North Dakota, Claudia Gullickson, gave a "behind the scenes" account of the Miss America Contest at our first meeting on October 21. The second meeting was held November 18, with Mr. Leonard Egstrom, Deaconess Hospital Administrator, as our guest speaker. We were very pleased to have Mrs. J. M. Van

der Linde, state president, as our guest at the February 18 meeting. Her interesting and informative talk on the Forand bill inspired many of us to write our congressmen. Our auxiliary also wrote resolutions opposing the bill and sent them to our senators, representatives, and Representative Mills. Mrs. Donald Van Keuren, soprano soloist, was also on the program for the evening of February 18. At our last meeting on March 17, Mrs. E. L. Grinnell, one of our members, gave a wonderful talk on her recent trip to Hawaii.

Our members have assisted the Grand Forks District Cancer Committee in assembling kits for cancer detection and have spoken to the Medical Students' Wives Club and opened their homes for its meetings. We have taken an active part in community projects, such as St. Michael's and Deaconess hospital auxiliaries, Red Cross, church groups, and other organizations.

One of our projects this year was the AAPS essay contest for high school students in our district. The Grand Forks District Medical Society contributed a first prize of \$25, second of \$15, and third of \$10. We were delighted to hear that the 2 essays submitted from our district placed first and third in the state. Our chairman wrote each high school in the district and arranged for a good deal of publicity through the *Grand Forks Herald*.

The Medical Student Loan Fund was another important project of the year. A dinner dance was held on March 25 at the Grand Forks Air Force Officer's Club. The net proceeds were \$375.20. Our auxiliary also voted to donate the \$30 not used by delegates to the state convention in 1959 and the \$15 which was the difference in the cost of making our programs this year rather than having them printed. Our total contribution to the Student Loan Fund was \$420.20.

All of the AMEF Christmas cards sent to our auxiliary and 104 sheets and envelopes of the AMEF stationery were sold, netting \$35.60. There were 8 memorial contributions totaling \$55. Therefore, we were able to send \$90.60 to AMEF. Our Bulletin chairman reported only 2 subscriptions.

All of the doctors' wives from the Grand Forks Air Force Base were invited to attend our meetings.

We were saddened by the death during the past year of one of our members, Mrs. Margaret T. Mulligan.

The following officers were elected for 1960-1961: Mrs. W. P. Keig, president; Mrs. R. E. Mahowald, vice-president; Mrs. Rodney G. Clark, secretary; and Mrs. Louis Silverman, treasurer.

MRS. EDGAR A. HAUNZ, President

Auxiliary President's Report—Fourth District

The Northwest District held 3 evening meetings this year in the homes of its members, 1 luncheon meeting, and 1 joint dinner meeting with the Medical Society. One of our members, Mrs. J. S. McArdle, with firsthand knowledge of the children's diabetic camp near Grand Forks, spoke on Camp Sionx at our first meeting. As a result of her talk, which stressed the financial needs of the camp, the auxiliary decided to make an annual contribution of \$50 to Camp Sionx.

We were pleased to have our state president, Mrs. Van der Linde, visit us in October and bring news of the national convention at Atlantic City. She gave us encouragement and incentive to work on the many projects of the national auxiliary.

The instructor for driver education at Minot Model High School spoke to us in January on safe driving. Following this meeting, members underwent individual half-hour checkups on their driving.

Our fourth meeting was held at the beginning of Cancer Month, and Dr. Gale Richardson, pathologist at one of our hospitals, showed us the films "Time and Two Women" and "Self Examination of the Breast." Members discussed questions with him pertaining to these films.

At our fifth meeting, held jointly with the Northwest District Medical Society, Dr. V. J. Fiseher showed slides of his world mission trip taken last summer.

Printed yearbooks were issued for the first time this year, and new copies of our recently revised bylaws were also issued. The paramedical careers chairman sent copies of the scholarship listings that were compiled last year to 20 schools in the area and asked that these be placed in the school libraries for ready reference. It was again decided that each member would make a contribution of \$10 toward the Student Loan Fund in lieu of a project. We expect to total \$300 or more by convention time in Student Loan contributions.

We awarded prizes to winners of our district essay contest of \$25, \$15, and \$10 for first, second, and third place winners. The Northwest District Medical Society contributed \$25, and the remaining \$25 was donated from our treasury. Publicity was sent in to the local paper prior to the contest and again following the awards. The winners were introduced at our March meeting and presented with their prize checks before the auxiliary.

A policy of contributing a \$10 memorial gift to AMEF was adopted this year. We contributed \$30 in contributions to AMEF from our treasury. Our quota of note paper was sold to members by the AMEF chairman, making a further contribution of \$50.

We have continued to cover subscriptions to *Today's Health* in our annual dues. In response to the president's recent plea to place copies of *Today's Health* in the hands of all teachers, a committee will see that member's copies are placed regularly in each school and college and given to instructors who have indicated interest in personal copies.

Members brought in occupational therapy material for the state hospital as suggested by the state mental health chairman and these will be sent to Jamestown.

Our legislation chairman and committee have worked diligently in the past two months in sending resolutions, telegrams, and letters to Washington concerning the Forand bill. They have also assumed a large share of the work in the local survey on the aging.

Our membership totals 38. We were pleased with the regular attendance and splendid cooperation from our Air Force Hospital wives in many of our projects this year.

MRS. LORMAN L. HOOPES, President

Auxiliary President's Report—Sixth District

The Sixth District Medical Auxiliary held 4 dinner meetings this year. The first meeting on October 20, 1959, was held at the Petroleum Room in the Prince Hotel in Bismarck. Mrs. H. Milton Berg showed slides of her very interesting "Round-the-World" trip. Also, to acquaint the new members with the aims and projects of the auxiliary, each chairman spoke on the duties and functions of her respective committee.

The second meeting was held in December at the Prince Hotel. Miss Christine Wassberg, a native of Sweden and a student at Bismarck Junior College, spoke on "Christmas in Sweden." A short business meeting followed. The auxiliary voted to send in a donation to "Open Your Heart Fund."

The third meeting was held in February 1960 at the Municipal Country Club. Our state president, Mrs. J. M. Van der Linde was our honored guest and speaker. She spoke on the highlights of the Chicago conference and the Atlantic City convention and also on the Forand bill. She stressed the importance of our 100 per cent participation in fighting this type of legislation. During the dinner hour, 2 instrumental groups from the Bismarck High School played several selections.

Our fourth meeting was held in April at the Petroleum Room at the Prince Hotel. Dr. Baker, the psychiatrist in Bismarck, spoke on the "History of Psychiatry" and "What the Clinic Attempts to Do for the Patient." The following officers were elected for the coming year: president, Mrs. R. H. Waldschmidt; vice-president, Mrs. Roger Berg; secretary, Mrs. M. A. K. Lommen; and treasurer, Mrs. Robert Kling.

We have 62 members in our auxiliary. We sent \$600 to the Student Loan Fund and \$118 to the AMEF through the sale of Christmas cards and the note paper. *Today's Health* subscriptions numbered 6 and *Bulletin* subscriptions 24. The AAPS essay contest response was poor. No prizes were awarded this year; however, the prize money has been increased for next year.

One of our main projects this year was our campaign against the Forand bill. A resolution made up by our legislative chairman, Mrs. Tudor, was sent to the House Ways and Means Committee and the congressmen in Washington. Also, the members wrote letters and had many of their friends write to the congressmen opposing this bill.

Many of our members are very active in various community and hospital activities.

The officers this year were: vice-president, Mrs. Waldschmidt; secretary, Mrs. Roger Berg; and treasurer, Mrs. M. A. K. Lommen. Mrs. Heffron was councillor.

I wish to thank the officers, the committee chairmen, and the members of this district for their splendid cooperation.

MRS. PHILLIP O. DAHL, President

Auxiliary President's Report—Seventh District

It is with pleasure that I write a résumé of Stutsman County Medical Auxiliary activities for 1959-1960.

Our first meeting was an autumn tea at my home. Pennants with auxiliary committee labels were arranged on 4 tables. We used our own members as resource people, and many facets of each of our duties were discussed during our coffee hour. After the coffee hour, the moderators summarized the discussion for each table. We formulated a constitution for our Future Nurses Club, and decided community service, AMEF, and legislation would be our priority projects.

Mrs. John Jestadt was our hostess at a Christmas buffet dinner. Mrs. T. E. Pederson, Mrs. C. W. Hogan, and Mrs. John Swenson presented a model program, patterned after the Dale Carnegie meetings, on "Our Greatest Contribution of Time and Service to our Community." It was a very stimulating program, and we all participated enthusiastically. Again, we gave baskets of food and clothing to 2 needy families.

Our state president, Mrs. Van der Linde, did a magnificent job for us at our February dinner meeting in handling the precarious legislative threat to medicine in the form of the Forand bill. She encouraged us to write our congressman, and the follow-up response was excellent. Mrs. E. A. Haunz, Grand Forks, was our guest.

To climax our year, Mrs. P. G. Arzt was honored at a tea in a French setting held at the home of Mrs. R. S.

Woodward. An AMEF memorial card was presented to Mrs. Arzt in memory of Dr. Arzt. Officers and delegates to convention were elected; future hopes and plans for future nurses were described by Mrs. Thakor, advisor; and State Hospital birthday party plans were discussed.

Through the combined efforts of our auxiliary, we have had tremendous interest in our Future Nurses Club. Mrs. Thakor and I were advisors, and we feel that some of our successful programs have been as follows: (1) civil defense films on rescue breathing, civil defense, nerve gas, and navy first aid; (2) tour of Crippled Children's School and speech by the physical therapist; (3) assisting Crippled Children's School with Christmas shopping, dyeing Easter Eggs, reading to children, and playing games with them; (4) assisting with immunization clinics; and (5) assisting with civil defense disaster.

We obtained our national charter for future nurses.

Mrs. J. W. JANSONIUS, President

Mrs. Jansonius then presented AMEF awards to Devils Lake, Minot, and Bismarck in recognition of their contributions from the sale of note paper.

Auxiliary President's Report—Eighth District

The auxiliary to the Kotana Medical Society meets annually when the doctors convene for their dinner meeting.

We now have 17 members and have donated \$100 to the Student Loan Fund and \$10 to AMEF. Dues are \$10.50.

All members participate in the local drives for the heart fund, polio, cancer, and so forth, besides the work specified for 2 hospital auxiliaries.

Mrs. J. P. CRAVEN, President

Auxiliary President's Report—Ninth District

The Southwestern District Medical Auxiliary held 4 business meetings during the year. They were entertained by their husbands at a Christmas smorgasbord at their December meeting.

The President, Mrs. Richard Raasch, and the vice-president, Mrs. Robert Hankins, attended the executive board meeting in Jamestown in October.

The main projects of the year were the AAPS contest and the Future Nurses Club of the St. Joseph's Hospital in Dickinson, directed by Mrs. R. W. Rodgers and Mrs. Robert Gilliland, and the Medicorps of Mott, under the direction of Mrs. Robert Hankins.

The Medicorps is the first club exploring careers in health in North Dakota to receive a Future Nurses Club national charter. The charter was granted by the National League for Nursing, New York.

There were 3 entries to the AAPS contest from the Southwestern District. First prize of \$25 was given to LaVonn Boehm of Mott, second prize of \$15 was given to Marie Jan Wehri of Glen Ullin, and third prize of \$10 went to Lorilee Mayer of Richardson.

Contributions of \$50 were given to the Medical Student Loan Fund and the AMEF.

The sum of \$50 was allotted for the Future Nurses Club and Medicorps members for pins, which will be awarded to girls earning the necessary points at a tea given in May by the medical auxiliary, and for necessary expenses for the 2 clubs during the coming year.

Funds were also allotted to purchase a necessary piece of equipment for the classroom of St. Joseph's School of Practical Nursing, the amount and type of equipment to be decided after learning the needs of the school.

Mrs. Henry Slominski, legislative chairman, wrote a letter to Senator Young placing the Southwestern District

on record as opposing the Forand bill. Each individual auxiliary member was urged to send a personal letter or telegram also.

The Southwestern District did not send a delegate to the meeting held in Fargo on January 30.

Mrs. J. M. Van der Linde state auxiliary president, was invited to our April 6 meeting but was unable to attend because of her busy schedule.

The Southwestern District has 26 members. The new officers for the 1960-1961 year are: Mrs. Robert Hankins, Mott, president; Mrs. Norman Ordahl, Dickinson, vice-president; and Mrs. William Buckingham, Elgin, secretary-treasurer.

MRS. RICHARD F. RAASCH, President

Auxiliary President's Report—Tenth District

When the Traill-Steele Medical Auxiliary was organized, members decided that it would be a social group and would meet 4 times a year at the time our husbands met. This has been the plan carried out by our organization over the years.

We have a membership of 9 and make what personal contributions we can to AMEF and the Student Loan Fund.

MRS. D. N. MERGERS, President

Motion was made by Mrs. M. M. Heffron that the district reports be accepted. Motion was carried.

Upon recommendation by Mrs. Van der Linde, Mrs. Jansonius moved that a list of duties be prepared in calendar form and sent to all officers, chairmen, district presidents, and councillors. Motion was carried.

Mrs. Baumgartner reported that her supply of *Handbooks* was low. It was recommended that she order 25 copies of the *Handbook*.

Mrs. Van der Linde read Dr. Thomas E. Pederson's recommendation that local districts form committees on aging. Motion was made by Mrs. Longmire that such committees be formed. Motion was carried.

Motion was made by Mrs. M. E. Nugent that the first issue of *News, Views, and Cues* be sent to all eligible doctors' wives and that later issues be sent only to members of the auxiliary. Motion was carried.

Motion was made by Mrs. H. L. Kernott, Jr., that, in Bylaws article 9, section 3, the word "September" be deleted and replaced by "in the fall." Motion was carried.

Motion was made by Mrs. M. M. Heffron that the presentation of the Student Loan Fund check be publicized by newspaper stories and pictures. Motion was carried.

Motion was made by Mrs. Jansonius that each district plan its own project to raise money for the AMEF. Motion was carried.

Mrs. H. L. Kernott, Jr., moved that we allow \$25 for our delegate to the National Convention to cover the cost of meals and that we allow \$90 to the Student American Medical Association Auxiliary to assist it in sending a delegate to its national convention. Motion was carried.

A committee appointed by Mrs. Van der Linde at the preconvention board meeting composed of Mrs. Jansonius, Mrs. Longmire, Mrs. Waldschmidt, and Mrs. Cardy recommended that the state be divided into 4 convention areas: (1) Williston, Devils Lake, Minot, and Rugby; (2) Jamestown, Valley City, and Fargo; (3) Grand Forks and Traill-Steele; (4) Dickinson and Bismarck. In each area, the smaller cities would assist the host city with convention arrangements. The committee also recommended that the president of the assisting districts be invited to the convention planning meeting.

Mrs. Gertson moved that the committee's recommendations be adopted. Motion was carried.

Motion was made by Mrs. Kermott that we recess for lunch.

In a setting of spring garden beauty, a most enjoyable luncheon was held at the YWCA Banquet Room on May 2, with Mrs. R. W. McLean presiding. Mrs. McLean introduced the following who were seated at the head table: Mrs. V. J. Fischer, Mrs. J. W. Fawcett, Mrs. J. W. Jansonius, Mrs. C. M. Lund, Mrs. L. T. Longmire, Mrs. S. C. Bacheller, Mrs. J. M. Van der Linde, Dr. C. M. Lund, Mrs. R. L. McFadden, Mrs. C. L. Smith, Mrs. C. J. Baumgartner, and Mrs. Charles Graham.

A gracious welcome was given by Mrs. Charles Graham of Grand Forks. She presented Mrs. Philip Andrews, who announced the program would consist of several tap and ballet dances. Mrs. Graham introduced Mrs. J. D. Cardy, convention chairman.

Mrs. McLean introduced Dr. C. M. Lund, president-elect of the North Dakota State Medical Association, who pointed out that problems for doctors have increased in the last twenty-five years. He cited the Forand bill as an example of future problems. He stated if this bill is passed, it would be followed by others giving medical care to all under Social Security. He suggested that each doctor and his wife should have 10 lay friends well informed on medical legislation problems. He stressed the need for better public relations and asked the help of the auxiliary in promoting them.

The second business session reconvened at the YWCA on Monday, May 2, 1960, at 2:30 P.M. Mrs. J. M. Van der Linde, president, called the meeting to order.

Mrs. Van der Linde asked for nominations for delegates to the national convention. As there was no one present who planned to attend the national convention, it was decided to bring up the matter of delegates again at the dinner and at the brunch. Mrs. Cardy announced there were 95 registrants, 12 delegates, and 3 guests.

Mrs. Keller moved that the auxiliary send a card of condolence to Mrs. Halliday on the death of Dr. Halliday, former state president. Motion was carried.

Mrs. Grinnell asked for a vote of thanks to Mrs. Van der Linde for her fine work as president of the auxiliary during the past year.

The Ryan Hotel was the scene for an informal banquet on Monday, May 2, at 6:30 P.M. Decorations carried out the theme of the convention "Birds of a Feather Flock Together." Mrs. E. A. Haunz, third district president, presided.

Speaker for the evening was Mrs. Stephen Bacheller of Enderlin, regional vice-president of the national auxiliary. Mrs. Bacheller started her address by citing some of the changes that our medical auxiliary has undergone. She stated that in spite of our increased membership, fewer than 50 per cent of our potential members belonged. She mentioned that we were still being urged to subscribe to the *Bulletin*.

She felt that we should have a member in each district acting as legislation chairman to lead the fight against creeping socialism, which has become galloping socialism. She called attention to the Murray-Wagner Bill of 1943 and the Forand bill and stated that, if we had federal aid to medicine, private practice of medicine would end. "If you can socialize medicine, you can control the doctors; you can control the people," she stated.

She felt that we should make it very clear that we were in favor of giving aid to those who were incapable of taking care of themselves. We could do this through our state welfare association. "Anything given to those

who have not earned it must be taken from those who have earned it. Democracy ceases when money is taken from those who have worked and saved," she stated.

She believes that if we socialize medicine, everything will become socialized. She quoted that of a group of people interviewed, 80 per cent felt that their doctors had given good service at a reasonable price. She felt that too many people believe what they read rather than what they have experienced, and she felt very strongly that the doctors' story should be heard.

A delightful brunch was held at the Grand Forks Country Club on Tuesday, May 3, 1960 at 10:30 A.M. Mrs. J. D. Cardy, convention chairman, presided.

The following officers were installed by Mrs. Stephen Bacheller: Mrs. R. W. McLean, president; Mrs. L. T. Longmire, president-elect; Mrs. John Jansonius, first vice-president; Mrs. R. H. Waldschmidt, second vice-president; Mrs. Clyde Smith, secretary; and Mrs. Carl Baumgartner, treasurer. Mes. Waldschmidt, Jansonius, Smith, and Baumgartner were represented by proxies at the installation ceremony.

The past president's pin was presented by Mrs. J. D. Cardy to Mrs. J. M. Van der Linde, who gave a short talk and was given a standing ovation. The president's pin was presented to Mrs. McLean by Mrs. Bacheller. In her acceptance speech, Mrs. McLean asked for our cooperation and hoped our confidence in her would be well placed.

Drawings were held for 3 door prizes. Mrs. Paul Lundquist then introduced the program, consisting of Menotti's short opera, "The Telephone," presented by University of North Dakota faculty members.

Motion was made for convention adjournment.

Postconvention Minutes

The meeting was called to order by Mrs. McLean. She announced she had made the following appointments:

Organization—Mrs. L. T. Longmire

Program—Mrs. J. W. Jansonius

Civil defense—Mrs. E. A. Haunz

Nominating—Mrs. J. M. Van der Linde

Publicity—Mrs. M. M. Heffron

Legislation—Mrs. L. G. Pray

Bulletin—Mrs. A. G. Sathe

Historian—Mrs. O. M. DeMouilly

AMEF—Mrs. B. A. Mazur

Parliamentarian—Mrs. T. Q. Benson

Mental Health—Mrs. R. L. McFadden

Paramedical careers—Mrs. Robert Schoregge

Bylaws—Mrs. V. J. Fischer

Safety—Mrs. John Fawcett

AAPS—Mrs. Ted Keller

Community service—Mrs. Mack Traynor, Jr.

Official Publications—Managing editor, Mrs. J. H. Mahoney; business and circulation manager, Mrs. Charles Arneson; and co-editor, Mrs. Robert Hankins

Finance—Mrs. E. J. Larson, chairman; Mrs. Carl Baumgartner, Mrs. W. L. Macaulay, Mrs. L. E. Wold, and Mrs. William Keig

Student loan fund—Mrs. J. A. Sandmeyer, chairman; Mrs. G. R. Richardson, Mrs. J. M. Keller, Mrs. R. D. Nierling, and Mrs. Ralph Leigh

Liaison Officer to SAMA—Mrs. J. E. Cardy

Reading committee—Mrs. Clyde Smith, chairman; Mrs. M. M. Heffron, Mrs. C. A. Arneson, and Mrs. Roger Berg

Special committee on aging—Mrs. D. L. Kohl

Mrs. McLean appointed Mrs. E. J. Larson and Mrs. R. D. Schoregge to audit the books.

The following were elected to the Nominating Committee: Mrs. J. M. Van der Linde, chairman; Mrs. G. G. Thorgrimson, Mrs. Marvin Geib, Mrs. Clifford Peters, and Mrs. L. L. Hoopes. Alternates are Mrs. R. W. Rodgers, Mrs. M. A. Norum, Mrs. R. D. Schoregge, and Mrs. Samuel Shea.

Mrs. McLean explained the calendar of duties, which

will be compiled and sent to all state officers, chairmen of standing and special committees, district presidents, and councillors.

Motion was made to adjourn.

District Presidents

First District Mrs. Calvin Fercho, 1747 S. 7th St., Fargo
 Second District Mrs. J. A. Terlecki, Minnewakan
 Third District Mrs. W. P. Keig, 2320 7th Ave. N., Grand Forks
 Fourth District Mrs. Gale R. Richardson, 12 10th St. S.W., Minot
 Fifth District members-at-large
 Sixth District Mrs. R. H. Waldschmidt, 600 N. Washington, Bismarck
 Seventh District Mrs. E. O. Hieb, 300 6th Ave. N.E., Jamestown

Eighth District Mrs. John Keller, 910 4th Ave. E., Williston
 Ninth District Mrs. Robert Hankins, Mott
 Tenth District Mrs. D. N. Mergens, Hillsboro

District Councillors

First District Mrs. John Bond, 516 N. 13th St., Fargo
 Second District Mrs. L. T. Longmire, 810 6th St., Devils Lake
 Third District Mrs. P. H. Woutat, 1205 Lincoln Drive, Grand Forks
 Fourth District Mrs. V. J. Fischer, 303 8th Ave. S.E., Minot
 Sixth District Mrs. M. M. Heffron, 320 Ave B West, Bismarck
 Seventh District Mrs. R. L. McFadden, 910 3rd Ave. N.E., Jamestown
 Ninth District Mrs. L. H. Reichert, 543 1st Ave W., Dickinson
 Tenth District Mrs. Keigh C. Vandergon, Portland

WOMAN'S AUXILIARY TO THE NORTH DAKOTA STATE MEDICAL ASSOCIATION 1959 MEMBERSHIP ROSTER

Ahlness, Mrs. Paul	Bowman	Chlmer, Mrs. A. E., Jr.	101 Reeves Court, Grand Forks
Amidon, Mrs. B. F.	1701 9th St. S., Fargo	Dahl, Mrs. Phillip O.	1111 S. Highland Acres Rd., Bismarck
Amstutz, Mrs. Kenneth	505 9th Ave. S.E., Minot		
Anderson, Mrs. F. E.	Underwood	Dailey, Mrs. W. C.	1812 Belmont Rd., Grand Forks
Andrews, Mrs. Philip	1905 Chestnut, Grand Forks	Darrow, Mrs. K. E.	716 S. 8th St., Fargo
Armstrong, Mrs. W. B.	1710 S. 8th St., Fargo	DeCesare, Mrs. F. A.	1401 S. 9th St., Fargo
Arneson, Mrs. A. O.	419½ S. 5th St., Grand Forks	DeLamo, Mrs. Robert	Northwood
Arneson, Mrs. Charles A.	714 2nd St., Bismarck	DeMonilly, Mrs. Oliver M.	1715 Ave. E East, Bismarck
Arzt, Mrs. Phillip G.	502 4th Ave. S.E., Jamestown	Dexine, Mrs. John L.	7 Airview, Minot
Ayash, Mrs. John J.	121 7th Ave. S.E., Minot	Dillard, Mrs. J. R.	620 S. 8th St., Fargo
Bachieller, Mrs. S. C.	Enderlin	Diven, Mrs. William L.	119 Ave. B West, Bismarck
Baker, Mrs. Cecil G.	821 N. 1st St., Bismarck	Dodds, Mrs. C. A.	1602 S. 10th St., Fargo
Baumgartner, Mrs. Carl J.	615 N. Washington, Bismarck	Doss, Mrs. D. R.	419 23rd Ave. S., Grand Forks
Barnard, Mrs. Donald M.	1212 S. 7th St., Fargo	Dunkart, Mrs. C. R.	208 4th Ave. W., Dickinson
Beall, Mrs. John A.	419 4th Ave. S.E., Jamestown	Dunkart, Mrs. Ralph	S.W. of City, Dickinson
Beithon, Mrs. E. J.	129 N. 5th St., Wahpeton	Ellis, Mrs. Gordon E.	602 14th St. W., Williston
Benson, Mrs. Theodore Q.	1524 Walnut St., Grand Forks	Elsworth, Mrs. John Nelson	207 14th Ave. Drive N.E., Jamestown
Benwell, Mrs. Harry D.	625 S. 3rd St., Grand Forks	Engstrom, Mrs. Perry N.	807 N. 2nd St., Wahpeton
Berg, Mrs. H. Milton	214 Ave. A West, Bismarck	Erenfeld, Mrs. Fred R.	616 Lincoln Ave., Minot
Berg, Mrs. Roger M.	219 Ave. B West, Bismarck	Eriksen, Mrs. Johan A.	815 Ave. C West, Bismarck
Berthman, Mrs. Herman J.	Linton	Evans, Mrs. Harold	1024 Reeves Drive, Grand Forks
Boerth, Mrs. E. H.	825 Griffin St., Bismarck	Ewert, Mrs. Artnr O.	Goodrich
Bond, Mrs. J. H.	516 S. 13th St., Fargo	Fawcett, Mrs. John C.	1125 5th St., Devils Lake
Borland, Mrs. V. G.	1511 S. 9th St., Fargo	Fawcett, Mrs. Robert M.	12th Ave., Devils Lake
Borrud, Mrs. Chester C.	729 16th St. W., Williston	Fennell, Mrs. William L.	Crosby
Bowen, Mrs. Jesse	221 7th Ave. W., Dickinson	Fercho, Mrs. Calvin	1747 S. 7th St., Fargo
Breslich, Mrs. Paul J.	818 4th St. S.E., Minot	Fischer, Mrs. V. J.	303 8th Ave. S.E., Minot
Brink, Mrs. Norvel O.	212 Ave. F. West, Bismarck	Fjelde, Mrs. J. N.	1526 S. 8th St., Fargo
Buckingham, Mrs. Tracy W.	1030 N. 5th St., Bismarck	Fortin, Mrs. H. J.	1440 8th St. S., Fargo
Buckingham, Mrs. William	Elgin	Foster, Mrs. Keith	734 8th Ave. W., Dickinson
Burton, Mrs. P. H.	415 8th St. S., Fargo	Fox, Mrs. William R.	315 Foster Ave., Rugby
Cameron, Mrs. Angus L.	318 8th Ave. S.E., Minot	Freise, Mrs. Paul W.	831 Mandan St., Bismarck
Cameron, Mrs. A. Malcolm	412 25th St. N.W., Minot	Fritzell, Mrs. K. E.	1120 Cottonwood, Grand Forks
Cardy, Mrs. James D.	1110 Reeves Drive, Grand Forks	Gache, Mrs. Otto C.	New Salem
Cartwright, Mrs. John T.	1110 S. Highland Acres Drive, Bismarck	Gammell, Mrs. Robert T.	Kennmare
		Garrett, Mrs. W. G.	1207 N. Washington, Bismarck
Chase, Mrs. Gilman	Mayville	Garrison, Mrs. M. W.	612 Mt. Curve Ave., Minot
Chernasek, Mrs. Sam	138 1st Ave. W., Dickinson	Geib, Mrs. M. J.	1219 4th Ave. S., Moorhead
Christianson, Mrs. G.	717 6th St. N.E., Valley City	Gertson, Mrs. G. D.	511 S. 5th St., Grand Forks
Christoferson, Mrs. Lee A.	1307 S. 6th St., Fargo	Gillam, Mrs. John S.	1433 S. 7th St., Fargo
Christu, Mrs. C. N.	1509 5th Ave. S., Fargo	Gilliland, Mrs. Robert	446 1st Ave. W., Dickinson
Clark, Mrs. R. G.	511 18th Ave. S., Grand Forks	Gilsdorf, Mrs. Amos	870 4th Ave., Dickinson
Clayburgh, Mrs. B. J.	729 Reeves Drive, Grand Forks	Giltner, Mrs. Lloyd A.	1000 4th Ave. N.W., Minot
Cleary, Mrs. Joseph W.	104 Seminole Ave., Bismarck	Girard, Mrs. B. A.	Beulah
Cook, Mrs. Stuart J.	Rolette	Gochl, Mrs. R. O.	1015 Reeves Drive, Grand Forks
Corbett, Mrs. Connor A.	316 7th St., Devils Lake	Goff, Mrs. John R.	1411 S. 8th St., Fargo
Corbus, Mrs. B. C.	1257 N. 4th St., Fargo	Goltz, Mrs. N. F.	804 S. 8th St., Fargo
Coultrip, Mrs. R. L.	Box 87, McVile	Goodman, Mrs. Edward	Napoleon
Craven, Mrs. John P.	403 3rd Ave. E., Williston	Goodman, Mrs. Robert	Powers Lake
Craven, Mrs. Joseph D.	915 W. 2nd Ave., Williston	Goughnour, Mrs. Myron W.	1310 N. 2nd St., Bismarck

Goven, Mrs. John W.	912 6th Ave. N.E., Valley City	Kuplis, Mrs. Haralds	Turtle Lake
Gozum, Mrs. Ekrem	1715 4th St. S.W., Minot	La Fleur, Mrs. Harold A.	Mayville
Graham, Mrs. Charles	923 Almonte, Grand Forks	Lampert, Mrs. Max T.	101 10th St. N.W., Minot
Graham, Mrs. J. H.	1125 Reeves Drive, Grand Forks	Lancaster, Mrs. W. E. G.	1332 N. 5th St., Fargo
Grinnell, Mrs. E. L.	1207 Lincoln Drive, Grand Forks	Landa, Mrs. Marshall	1720 S. 8th St., Fargo
Gregware, Mrs. Peter R.	1107 S. Highland Acres Rd., Bismarck	Landry, Mrs. L. H.	Wallhalla
Guloien, Mrs. Hans	45 5th Ave. W., Dickinson	Larsen, Mrs. Harlan	1005 5th Ave. W., Dickinson
Gumper, Mrs. Arnold	7 E. 4th St., Dickinson	Larson, Mrs. Ernest J.	321 2nd Ave. S.E., Jamestown
Gustafson, Mrs. Maynard B.	1201 S. 8th St., Fargo	Larson, Mrs. G. A.	1538 S. 9th St., Fargo
Hagen, Mrs. Edward J.	904 2nd Ave. E., Williston	Larson, Mrs. L. W.	200 Tower Ave., Bismarck
Hagen, Mrs. Joan G.	410 2nd Ave. E., Williston	Lawson, Mrs. Mason G.	200 Ridgeway, Little Rock, Ark.
Hall, Mrs. C. H.	1748 S. 9th St., Fargo	Lazareck, Mrs. Isadore L.	1032 5th St., Devils Lake
Halliday, Mrs. David	Kenmare	LeBein, Mrs. W. E.	1353 5th St. N., Fargo
Hamilton, Mrs. C. A.	1212 N. Washington St., Bismarck	Leigh, Mrs. James A.	606 N. 3rd., East Grand Forks
Hanewald, Mrs. Walter	Richardton	Leigh, Mrs. Ralph E.	301 Park Ave., Grand Forks
Hankins, Mrs. Robert	Mott	Leigh, Mrs. Richard H.	1117 Lincoln Drive, Grand Forks
Hanson, Mrs. Harris D.	402 Ave. C West, Bismarck	LeMar, Mrs. J. D.	1324 N. 5th St., Fargo
Harwood, Mrs. T. H.	2704 Belmont Rd., Grand Forks	Levi, Mrs. Wesley E.	1215 1st St., Bismarck
Haugen, Mrs. C. O.	Box 436, Larimore	Levine, Mrs. Leo	1006 Cottonwood St., Grand Forks
Haunz, Mrs. Edgar A.	1029 Lincoln Drive, Grand Forks	Lewis, Mrs. T. H.	1502 S. 6th St., Fargo
Hawn, Mrs. Hugh W.	1325 N. 1st St., Fargo	Liebeler, Mrs. W. A.	2001 Chestnut, Grand Forks
Heffron, Mrs. Maurice M.	320 Ave. B West, Bismarck	Lindelow, Mrs. O. Victor	831 Crescent Lane, Bismarck
Heidorn, Mrs. G. H.	1127 Valley View Drive, Minot	Lindsay, Mrs. D. T.	1505 S. 11th St., Fargo
Heilman, Mrs. Charles	49 18th Ave. N., Fargo	Lipp, Mrs. George R.	502 Rosser Ave. W., Bismarck
Helenbolt, Mrs. K. S.	2216 S. 10th St., Grand Forks	Little, Mrs. James M.	Mayville
Helm, Mrs. Richard	1505 N. 5th St., Grand Forks	Little, Mrs. Roy C.	Mayville
Henderson, Mrs. Robert W.	1028 4th St., Bismarck	Lommen, Mrs. M. A. K.	831 Griffin St., Bismarck
Hendrickson, Mrs. George C.	2922 N. 7th St., Fargo	London, Mrs. Carl B.	506 Main St. S., Minot
Hetzler, Mrs. Arnold E.	602 Sixth Ave. N.W., Mandan	Long, Mrs. W. H.	1438 8th St. S., Fargo
Hieb, Mrs. Edwin O.	211 14th Ave. Drive N.E., Jamestown	Longmuir, Mrs. Lemuel T.	810 6th St., Devils Lake
Hill, Mrs. Simon	Regent	Lucy, Mrs. Robert	420 4th Ave. S.W., Jamestown
Hilts, Mrs. George H.	Cando	Lund, Mrs. Carrol M.	701 1st Ave. E., Williston
Hogan, Mrs. Clifford	303 10th Ave. N.E., Jamestown	Lytle, Mrs. F. T.	1306 1st St. N., Fargo
Holt, Mrs. George H.	214 2nd Ave. S.W., Jamestown	MacDonald, Mrs. A. C.	607 5th Ave. N.W., Valley City
Hoopes, Mrs. L. L.	118 9th Ave. S.E., Minot	MacDonald, Mrs. Neil A.	711 5th Ave. N.W., Valley City
Hordinsky, Mrs. Bohdanz	Drake	McArdle, Mrs. John S.	222 Souris Drive, Minot
Houghton, Mrs. J. F.	1707 S. 9th St., Fargo	McBane, Mrs. Robert D.	1106 4th St., Devils Lake
Hunter, Mrs. C. M.	1434 S. 6th St., Fargo	McCannel, Mrs. A. D.	505 Main St. S., Minot
Huntley, Mrs. W. B., Jr.	208 7th Ave. S.E., Minot	McCullough, Mrs. William F.	Bottineau
Hurly, Mrs. William C.	6 9th St. S.E., Minot	McDongall, Mrs. James R.	601-B Main St. S., Minot
Ivers, Mrs. George U.	1106 S. 10th St., Fargo	McFadden, Mrs. Robert L.	910 3rd Ave. N.W., Jamestown
Jacobson, Mrs. M. S.	Elgin	McLean, Mrs. Robert	Hillsboro
Jaehning, Mrs. David	823 N. 3rd St., Wahpeton	McLeod, Mrs. John	911 N. 22nd St., Grand Forks
James, Mrs. J. B.	1145 N. 10th St., Fargo	McPhail, Mrs. C. O.	Crosby
Jansonius, Mrs. John	609 4th Ave S.E., Jamestown	Macanley, Mrs. W. L.	1410 9th St. S., Fargo
Jensen, Mrs. August F.	1721 Belmont Rd., Grand Forks	Magill, Mrs. Gordon B.	1312 S. 7th St., Fargo
Jensen, Mrs. Warren	521 4th Ave N.W., Valley City	Magness, Mrs. John	1711 S. 6th St., Fargo
Jestadt, Mrs. John J.	419 15th St. N.E., Jamestown	Mahoney, Mrs. James H.	601 8th St., Devils Lake
Johnson, Mrs. Alan K.	1004 E. 4th St., Williston	Mahowald, Mrs. R. E.	606 S. 5th St., Grand Forks
Johnson, Mrs. Chris	819 W. 3rd St., Rugby	Marshall, Mrs. Robert	137½ S. 3rd St., Grand Forks
Johnson, Mrs. K. J.	216 Tower Ave., Bismarck	Mazur, Mrs. B. A.	1237 N. 3rd St., Fargo
Johnson, Mrs. M. J. E.	1020 N. Washington St., Bismarck	Melton, Mrs. Frank	1702 S. 7th St., Fargo
Johnson, Mrs. O. W.	422 W. 3rd St., Rugby	Mergens, Mrs. Daniel N.	Hillsboro
Johnson, Mrs. Paul L.	224 Ave. A West, Bismarck	Merrett, Mrs. J. P.	801 5th Ave. N.E., Valley City
Kahins, Mrs. Arnold	Washburn	Miles, Mrs. James V.	722 6th Ave. S.E., Jamestown
Keig, Mrs. William P.	2320 7th Ave. N., Grand Forks	Moore, Mrs. John H.	1114 Reeves Drive, Grand Forks
Keller, Mrs. Emil T.	Rugby	Nachtwey, Mrs. A. P.	115 5th Ave. W., Dickinson
Keller, Mrs. John	910 E. 4th Ave., Williston	Nazli, Mrs. Mehmet H.	316 18th Ave. S.W., Minot
Kermott, Mrs. L. Henry	200 7th Ave. S.E., Minot	Nelson, Mrs. Wallace W.	511 17th Ave. S., Grand Forks
Klein, Mrs. Clifford	117 3rd St. N.W., Valley City	Nelson, Mrs. William	1118 Reeves Drive, Grand Forks
Kling, Mrs. Robert R.	1414 Hanaford Ave., Bismarck	Nierling, Mrs. Richard D.	415 9th St. S.E., Jamestown
Knickerbocker, Mrs. Walter	Hettinger	Norum, Mrs. H. A.	801 South Drive, Fargo
Kohl, Mrs. Darwin L.	209 8th Ave. S.E., Minot	Nuessel, Mrs. Robert F.	815 Griffin St., Bismarck
Korwin, Mrs. Justin J.	701 1st Ave. E., Williston	Nugent, Mrs. Milton E.	302 W. Boulevard, Bismarck
		Oja, Mrs. R. Karl	Ashley
		Olson, Mrs. Burton G.	629 3rd St. S.E., Minot

Olson, Mrs. Donald L.	911 S. 8th St., Fargo	Smith, Mrs. Clyde L.	622 Raymond St., Bismarck
Orchard, Mrs. W. J.	Linton	Smith, Mrs. Oscar	519 1st Ave. W., Dickinson
Ordahl, Mrs. Norman	N.W. of City, Dickinson	Sorenson, Mrs. Alfred R.	114 6th St. S.E., Minot
Orr, Mrs. August C.	922 9th St., Bismarck	Sorenson, Mrs. Roger	101 9th St. S.E., Minot
Oster, Mrs. Ellis	Ellendale	Sorkness, Mrs. Joseph	318 3rd Ave. S.E., Jamestown
Owens, Mrs. P. L.	827 Griffin St., Bismarck	Spear, Mrs. A. E.	610 1st Ave. W., Dickinson
Painter, Mrs. Robert C.	1121 Belmont Rd., Grand Forks	Stafne, Mrs. William A.	1409 9th St. S., Fargo
	Cando	Stangebye, Mrs. T. L.	1620 Braman Ave., Bismarck
Palmer, Mrs. Dolson W.		Story, Mrs. R. D.	1315 S. 9th St., Fargo
Pederson, Mrs. Thomas E.	116 4th Ave. N.E., Jamestown	Stratte, Mrs. Joseph J.	109 4th Ave. S., Grand Forks
Perrin, Mrs. Edwin D.	520 Ave. A West., Bismarck	Strinden, Mrs. Dean R.	1717 8th Ave. W., Williston
Peters, Mrs. Clifford H.	805 Griffin St., Bismarck	Swanson, Mrs. J. G.	1220 S. 8th St., Fargo
Peterson, Mrs. Paul A.	2702 N. 8th St., Fargo	Swenson, Mrs. John A.	240 12th Ave. N.E., Jamestown
Pettit, Mrs. Sam	625 Reeves Drive, Grand Forks	Tarpley, Mrs. H. I.	511 Reeves Drive, Grand Forks
Pianka, Mrs. Wallace J.	Veterans Hospital Quarters, Fargo	Terlecki, Mrs. Jaroslaw	Minnewaukan
		Thakor, Mrs. Hushen	State Hospital, Jamestown
		Thom, Mrs. Robert	Bowman
Pierce, Mrs. W. B.	911 Ave. C West, Bismarck	Thompson, Mrs. George	421 S. 14th St., Fargo
Pierson, Mrs. R. Warren	1209 Mandan St., Bismarck	Thorgrimsen, Mrs. C. G.	1615 4th Ave. N., Grand Forks
Pile, Mrs. Duane F.	Crosby	Toomey, Mrs. Glen	Highway 20, Devils Lake
Pine, Mrs. Lonis Fabien	817 7th St., Devils Lake	Traynor, Mrs. Mack V.	1310 S. 9th St., Fargo
Poindexter, Mrs. M. H.	1350 S. 9th St., Fargo	Triggs, Mrs. Perry O.	1401 S. 12th St., Fargo
Porter, Mrs. Charles B.	1210 Chestnut, Grand Forks	Tindor, Mrs. Robert B.	714 Ave. C. West, Bismarck
Potter, Mrs. W. F.	2024 2nd Ave. N., Grand Forks	Turner, Mrs. Neville	LaMoure
Powers, Mrs. W. T.	1509 Walnut, Grand Forks	Uhner, Mrs. R. J.	1433 S. 12th St., Fargo
Prochaska, Mrs. L. J.	620 Reeves Drive, Grand Forks	Uthus, Mrs. Oliver	916 Central Ave. W., Minot
Pray, Mrs. L. C.	1701 S. 8th St., Fargo	Vaaler, Mrs. R. A.	1711 6th St. S.W., Minot
Rausch, Mrs. Richard F.	30 West 8th St., Dickinson	Vandergon, Mrs. Keith Gordon	Portland
Ramstad, Mrs. N. O.	824 4th St., Bismarck	Van der Linde, Mrs. John	1016 4th Ave. N.E., Jamestown
Reichert, Mrs. Donald	1019 5th Ave. W., Dickinson		Oakes
Reichelt, Mrs. L. H.	543 1st Ave. W., Dickinson	Van Honten, Mrs. Richard	
Richardson, Mrs. Gale	12 10th St. S.W., Minot	Vareo, Mrs. B. W.	701 29th Ave. N., Fargo
Rodgers, Mrs. R. W.	146 W. 6th St., Dickinson	Vigeland, Mrs. George N.	Rugby
Rogers, Mrs. Robert G.	1621 7th St. S., Fargo	Vonnegut, Mrs. Felix J.	Linton
Rosenberg, Mrs. Mervin	Northwood	Wakefield, Mrs. K. M.	Cooperstown
Rund, Mrs. John E.	2221 Chestnut, Grand Forks	Waldschmidt, Mrs. R. H.	600 Washington, Bismarck
Sahl, Mrs. Jens	310 9th St. N.W., Minot	Waldschmidt, Mrs. William D.	1241 S. Highland
Sandmeyer, Mrs. John A.	1005 Lanark, Grand Forks		Acres Rd., Bismarck
Sathe, Mrs. Andrew G.	718 15th St. W., Williston	Walker, Mrs. H. Charles	1701 Hillside Court, Williston
Schneider, Mrs. J. F.	1002 S. 13th St., Moorhead, Minn.	Wasdahl, Mrs. W. A.	611 N. 24th St., Grand Forks
Schoregge, Mrs. Charles W.	507 6th St., Bismarck	Webster, Mrs. William O.	823 S. 14th St., Fargo
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Book Reviews . . .

The Foot and Ankle

PHILIP LEWIN, M.D., 1959. Philadelphia: Lea & Febiger. 612 pages. Illustrated. \$14.00.

In this book, Dr. Lewin presents material related to the medical and surgical problems of the foot and ankle that is encyclopedic in its variety and scope and undoubtedly represents his broad experience and research into the literature over a period of many years. The student and practitioner interested in treatment of the foot and ankle can expand their general knowledge of this subject immensely by studying Dr. Lewin's "pearls" of information, presented in numerous short, one-sentence paragraphs. Though tedious and, at times, repetitious, they are nonetheless valuable guides from a practitioner of long experience. Unfortunately, the organization of the subject matter and its manner of presentation are extremely poor. After the regular chapters, an oversized appendix—71 pages—is inserted, representing afterthoughts; with more conscientious planning, these could have been included in their proper categories in the regular text.

The illustrations are numerous and of good quality, but, in many instances, the legends are confusing, and the illustrations lack proper correlation with material in the text. The illustration of Leo Mayer's peroneus longus transfer, for example, appears in the middle of a paragraph describing extensor hallucis longus transfer, and the appropriate text appears four pages later.

Dr. Lewin's presentation is at its best in describing the nonoperative treatment of static deformities and in outlining the multitudinous medical, neurologic, and vascular conditions which affect the foot and ankle directly or indirectly. Chapter 20, on "Disability Evaluation," is a model of concise presentation of valuable material and contains an excellent table for estimating disability of the foot and ankle. Surgical procedures, however, are usually merely listed, and the techniques are described without adequate reference to the proper indications for surgery. The Miller procedure for flat foot is presented without any reference to the indications favoring surgery over conservative care or the approximate age range of the candidates for surgery. Such an omission can lead to numerous unwarranted surgical procedures for pes planus on patients who could have been better treated by conservative means.

It is a pleasure to read Dr. Lewin's description of the bone stabilization procedures for poliomyelitis, for the author was acquainted with the men who developed these procedures, which have withstood the test of time and are now basic in the practice of orthopedic surgery. The tendon transfers presented, however, are in my opinion mainly of historic interest; in future texts they should be supplanted by more effective tendon transfer procedures, as presented in the more recent literature. It is not surprising that the majority of orthopedic surgeons become discouraged with tendon transfer about the foot and ankle when they are only offered, as in this book, transfer of the extensor hallucis longus or peroneus longus to replace anterior tibial paralysis and no transfer procedure to replace posterior tibial paralysis. Bickel and Moe's translocation of the peroneus longus tendon for replacement of triceps surae is unusually complicated.

It should be replaced by simple transfer of the peroneus longus to the os calcis.

It is discouraging to find Dr. Lewin recommending the subtalar bone block of Grice, which, although of some value, has recently reached the proportions of an orthopedic fad in the treatment of paralytic and static flat feet. In my opinion, the surgeon contemplating a subtalar bone block for treatment of severe valgus or pes planus following poliomyelitis should first read the important paper of Fried, Annon, and Hendel (Paralytic Valgus Deformity of the Ankle. *J. Bone & Joint Surg.* 39-A:921-932, 1957) describing transfer of the peroneus longus for replacement of the posterior tibia. For static pes planus, many operative procedures already well recognized, including some described in Dr. Lewin's book, are superior to the Grice bone block. In chapter 20, on "Gangrene of the Foot and Ankle," the author does not distinguish between sites of election applicable to traumatic amputations and those indicated for circulatory impairment. This omission is dangerous, because end-bearing stumps tend to break down and give trouble if there is circulatory impairment. Most surgeons consider the end-bearing stump contraindicated in amputations for circulatory impairment.

The reader is also advised to skip chapters 17 and 18, on "Fractures of the Ankle and Foot," in favor of other texts and monographs on fractures. Bonnin's *Injuries to the Ankle* (Grune & Stratton, 1950) is especially recommended for fractures of the ankle.

Despite my numerous criticisms, this book will find a welcome spot on my bookshelf and will be consulted to remind me of the numerous details of diagnosis and treatment, many of which I am prone to forget because of their rarity. Any practitioner treating foot conditions will find much of value, and the book should be especially valuable to the student, intern, or resident seeking a multitude of facts about the foot and ankle under one cover.

EUGENE H. CHAPMAN, M.D.
Provo, Utah

Readings on Race

STANLEY M. GARN, PH.D., 1959. Springfield, Ill.: Charles C Thomas. 279 pages. \$6.75.

This book provides an excellent opportunity for physicians to learn about recent trends in biology as applied to man. The study of race has been changing, both in direction and in methods of research. Dr. Garn points to a 1950 symposium, which included physical anthropologists and geneticists, as the turning point, although the basic principles had been established earlier.

The readings selected are investigations of natural selection, population size, and genetic drift as they affect changes in the frequency of genes. They illustrate the present concern with the processes of human evolution rather than with a classification of the products. A race is now considered as changing rather than fixed. Thus, it is no longer meaningful to think of fixed primary races, with all variations as admixtures. Furthermore, the number of races may be as few as 6 or as many as 30, depending upon the level at which the term is defined.

Considerable attention is given to the sickle-cell trait

as a prime example of balanced polymorphism. The distribution of this trait in Africa and other countries had presented many problems which now can be interpreted in terms of the recent finding that heterozygotes are relatively immune to malaria. The spread of malaria itself is related to the introduction of agricultural methods. This is an intriguing story of human disease as an important factor in determining the course of human evolution.

For some time, the frequencies of ABO and other blood group genes have been used to describe human races, under the assumption that these genes are adaptively neutral and thus suitable as markers. Recent evidence, however, has demonstrated associations between the ABO groups and certain diseases and also interactions between the ABO and Rh systems. Nevertheless, blood group data can be used to study admixture in recent populations, as illustrated by 2 papers estimating the amount of white admixture in the American Negro population and the average gene flow rate per generation.

Seven of the selections analyze differences in tolerance to cold and hot climates as related to such factors as body build, weight, basal metabolism, fat distribution, and nose shape. The brief paper by Wilber indicates the caution that must be used in interpreting such associations in terms of causal mechanisms. The principles discussed in these selections have important implications for those engaged in anatomic or physiologic research related to race.

Somewhat different interpretations might be given to a few points. The assumption that the recessive carrier of Morquio's syndrome is likely to have an advantage seems unnecessary. The general association between dark skin color and warm climate may have resulted from a selection for protective coloration rather than for heat tolerance.

The editor planned this as "more a case-book than a text-book, a book that includes the theorists' own words and the investigators' plans of action." A brief and helpful editorial interpretation introduces each section. The selected papers have just enough tables and graphs to give an adequate understanding of the data required for the generalizations. Over 350 additional references are cited. Most of the terms will be familiar to a physician, but the setting will be new and the perspective should be refreshing.

V. ELVING ANDERSON, M.D.
Minneapolis

Biochemistry of Blood in Health and Disease

I. NEWTON KUGELMASS, M.D., PH.D., 1959. *Springfield, Ill.: Charles C. Thomas. 508 pages. Illustrated. \$15.75.*

For this reviewer, a clinician who has had to glean the overwhelming developments in the field of internal medicine of the last generation by very selective and often inadequate coverage of the literature, this book on the biochemistry of blood is an excellent compilation of the facts in this field up to and including contributions of 1958. In general, if texts as a group are usually five years behind the current literature, Dr. Kugelmass has made a very thorough and inclusive review of the progress of biochemistry that is far ahead in the currency of the material. He, himself, has been quite active in the field since 1921. Scattered throughout the text are references to contributions made by Dr. Kugelmass to

the subjects of hypoglycemia, acid-base balance, serum and blood iron, relation of copper to microcytic anemia, the nature of bioflavonoids and their relation to vascular purpura, hemolysis, and, of course, blood coagulation. Often these contributions are particularly related to the pediatric field. Dr. Kugelmass, primarily a biochemist with a pediatric background, has, therefore, been able to condense a wide and diffuse field into a concise epitome. Because of the segmental and interdependent developments and contributions by so many workers in the field, many of the statements are documented by an excellent, comprehensive, current, and key bibliography.

The book is divided into 3 sections. The first covers well-established and the more recent data on the metabolism of proteins, carbohydrates, and fats. The charts, drawings, and tables are plentiful and instructive and summarize quickly much of the descriptive material such as metabolic pathways, cycles, and structures. Excellent correlation is made with clinical medicine. Areas of controversy are handled by documenting the opinions of the researchers. This is true particularly in the area of the lipids. It is of interest that Kugelmass made an original observation that a high fat diet increased blood coagulability in 1935. This observation was confirmed by Fullerton in 1956 but has been questioned by other authors.

The second part deals with water, electrolytes, acid-base balance, minerals, and the "regulatory compounds"—the vitamins, enzymes, and hormones. This section is also comprehensive, authoritative, and thoroughly readable. It is particularly helpful in the manner and approach of the relationship of clinical medicine. Newer data on the trace metals, manganese, cobalt, copper, and iodine, and on the bulk metal, magnesium, are presented.

The third portion of this text deals with the red cell, its function as a carrier of hemoglobin, its oxygen-carrying function, and its deficiency and surplus in the anemias; polycythemias; pigment disorders of heme and bile; and the hemoglobinopathies. The section also deals very comprehensively with blood clotting, anticoagulants, blood grouping, transfusion problems, the leukocytes, and immune bodies. The new coagulation factors and their interrelationships are discussed in a more understandable fashion. This reviewer, however, still believes that there is nothing as complex (and being made more so by more recent knowledge) than the problem of coagulation.

Several interesting appendices give the chemical units of measurements; the normal range of concentrations of an exhausting list of blood components; and abbreviations, symbols, and units. Of interest, too, is the fact that each chapter is preceded by a list of the pioneers in that particular sphere. For historic orientation, I found this very welcome.

Dr. Kugelmass in his preface states that the purpose of this book is "to present the newer knowledge of human blood in health and disease in the light of chemical research, clinical experience." This he has done with consummate skill through a thorough knowledge of the area and a truly academic interest in the work of the principal and key contributors in the field. It is a very concise, comprehensive, and excellent presentation of biochemistry clinically oriented so that it is highly recommended to the practicing physician and the medical student.

NATHANIEL G. BERK, M.D.
Philadelphia

(Continued on page 26A)

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BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, New York

Book Reviews . . . continued

Master Your Tensions and Enjoy Living Again

GEORGE S. STEVENSON, M.D., and HARRY MILT, 1959.
New York: Prentice Hall. 237 pages. \$4.95

Very real problems are inherent in producing a book such as this. Among them is the need to write with scientific and clinical validity in understandable, simple English; this is well done. Psychologic and psychiatric jargon is pleasantly absent. Moreover, to write a book on how to "master your tensions" and not fall into the trap of platitudinous description is no mean accomplishment.

The unique combination of Dr. George S. Stevenson, with his vast medical and scientific knowledge in the field of mental health, and the writing ability of Harry Milt, informed as he is in mental health matters, has produced a combination which is both trustworthy and readable. These authors are staff members of the National Association for Mental Health. The former is a national and international psychiatric consultant for the organization, and Mr. Milt is public relations director.

Perhaps some will quarrel with the near didactic style of direct conversation with the reader in terms of "you." Perhaps others will regret the too compact, and yet too diffused, last 4 chapters which suffer from the attempt at too much in too little space. However, all will appreciate the variety of possibilities presented for personal application of insights gained concerning "Tension—What It Is and Where It Comes From." This is accomplished without asking readers for intense introspection or implying that those who are facing problems with tensions are bordering on emotional disorganization or illness. In other words, this section is a healthy approach to persons who have within themselves potential for releasing the tensions inherent in everyday situations and relationships. Fear and anxiety are discussed and examined without leaving a residue of either. Moreover, reassurance may well be the end result of this presentation which recognizes fear and anxiety as among the daily components of living.

Perhaps the most difficult feat of this book is the development of an amazing number of situations and relationships illustrative of each of the discussed 8 "tension breakers." Titles of these tend to contribute to release of the tensions under discussion: "Talk it Out," "Escape for a While," "Take One Thing at a Time," "Get Rid of Anger," "Curb the Superman Urge," "Take a Positive Step," "Do Something for Somebody Else," and "Knock Down the Barbed Wire Fences." Here is the art of simple words combined with the wisdom of the behavioral sciences. Here in everyday language are living realities for the mentally healthy person and the desired end product for those who are troubled. Here are preventive measures which can be achieved. Here is a statement of the uncommon sense of science formulated into a pattern for the art of living. Clues to distinctive approaches to suit different personalities are abundant. Not once is it implied that everyone goes about realization of these basic principles of effective interpersonal relationships except in the way which is deemed best by the person to suit his need.

If one could wish for more in a book packed with

valid information on human behavior and yet presented in the rich economy of simple words, it would be upon the reality that tensions are to be expected, accepted, and recognized as basic motivation for continuous growth and development. Perhaps another "tension breaker" might have been added at just this point. Without problems, demanding situations, and challenges to potentialities, little stimulation would exist toward change in behavior or in situations. While this was not said, it was implied and in some instances stated in passing. Perhaps the nearest to it was presented in the 2 chapters on "What Love for Your Children Can Do for You" and "Discipline for Children—When and How Much?" which stress the need for responsibility and for the limitations of healthy discipline in the lives of children as well as adults.

No matter what their specialties, professions involved with persons and their problems will find in this book a volume which they may recommend with confidence. It is a "teacher" of effective handling of self and self with others rather than the too frequently attempted solver of all problems through a single prescription for behavior.

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Pathology

PETER A. HERBUT, M.D., 1959, second edition. Philadelphia: Lea & Febiger. 1,516 pages. Illustrated. \$18.50.

This book is added to the ever-growing list of recently published books on pathology, which are primarily directed toward students of medicine. Dr. Herbut's wealth of experience is immediately noted as he discusses the many pertinent aspects of the subject at hand. The author has added 288 pages and 213 photographs to this revised second edition and still offers an encyclopedic amount of information in a single volume. The most pleasant change has occurred at the beginning of each chapter, where the pathologic physiology concerning the organ or system therein discussed is briefly reviewed. In this way, the author has subtly interwoven morbid anatomy with clinical medicine and clinical pathology. He has wisely proportioned his time in doing this. Following these résumés, the pathology of each organ or system is fully discussed under these headings: congenital anomalies, degenerations, inflammations, physical disturbances, and tumors. The author has rigidly adhered to these headings throughout the entire book.

The discussion of the respiratory and genital tracts is very comprehensive and, in places, quite detailed. The pathology of the gastrointestinal system is also detailed, leaving little else to be described. The central nervous system is treated in the usual manner, without any single facet being outstanding. It is refreshing to note the presence of so many original and well-taken photographs, which add so much to the text and reflect the author's extensive experience.

Since the first edition, very little revision has occurred in the bibliography at the end of each chapter. The

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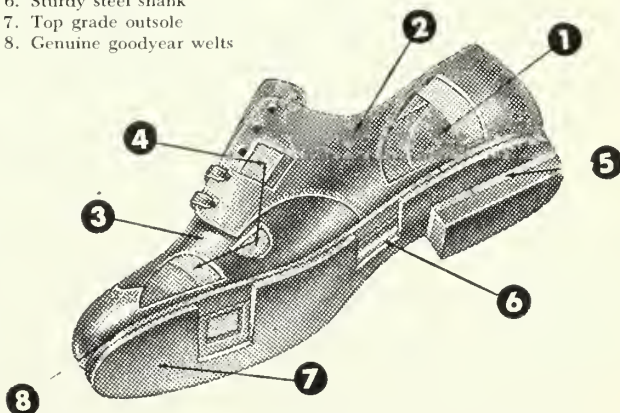


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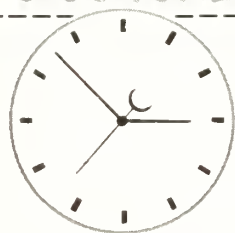
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¹Jorres, S. M.: *Unpublished test report from Pratt Diagnostic Clinic, New England Medical Center, Boston, Mass. (July, 1958)* NW-660



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BOOK REVIEWS

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references are old, and many leading articles are omitted. The paper is of good quality and the type is large and easy to read.

In summary, this is a good textbook on systemic pathology. It offers a good introduction into the subject and would acquaint the student or medical practitioner with the fundamentals of pathology.

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Longitudinal Studies of Child Personality

ALAN A. STONE, M.D., and GLORIS ONQUE, M.D., 1959.
Cambridge: Harvard University Press. 295 pages.
\$5.00.

This annotated bibliography contains 297 brief abstracts of studies published from 1923 to 1955 which used longitudinal methods to study the emotional and social development of children. Abstracts are arranged alphabetically by author and are cross-indexed for content. Within the very broad limits of a longitudinal method as defined by the authors, the bibliography is based on a thorough search of the literature and is presented in noncritical form.

The book is described as the first in a series of planned publications of a long-term, longitudinal study at the Yale University Child Study Center. As part of a bibliography serving a longitudinal project, perhaps the book served some useful purpose to those engaged in that particular research. But as a separate publication intended to be of some value to other professional workers, the book's worth is indeed doubtful. What group or what function could be served by this promiscuous collection of brief, uncritically presented abstracts is genuinely difficult to imagine.

A fundamental factor in the book's apparent failure to serve any public purpose is the peculiar decision to organize a bibliography in child development in terms of a broadly defined longitudinal method. Some types of research methods certainly are proper subjects for evaluation and study through presentation of assorted studies that have employed the method under scrutiny. But longitudinal methods encompass such vague and heterogeneous procedures as to render meaningless a major classification of literature based solely on those terms. To select studies which meet the criteria of a loosely defined method and vaguely defined content area and then to let all other factors vary randomly is to illuminate neither method nor content. If one were seeking information regarding longitudinal research methodology—or any methodology—this book certainly would not be helpful. If one were seeking information regarding a particular area, for example, psychologic reactions to surgery, certainly the search should not be organized in terms of any one type of methodology.

It would be wrong, however, to leave notice of this book on a harshly critical note. Whatever its defects, the publication deserves honorable mention as a reflection of a spirit too seldom evident in research on child personality development. Child psychiatry and child psychology have often been strangely divorced parents, each caring for the same children in ignorant and hostile denial of the other's worth. Here are two young psychiatrists who made a mighty effort to assemble the pertinent knowledge from each field before starting a major research effort. They chose the wrong method, in this writer's opinion, but their essential goal and good efforts deserve warm approval and encouragement.

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